

Noise Levels in the New Zealand Health Industry

by

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Abstract

The aim of this study was to investigate noise levels in the New Zealand health industry. The goal was to investigate the room acoustics and the characteristics of the noise sources along with noise exposure of health care workers, in New Zealand, in dental clinics and orthopaedic cast clinics and assess whether they are at risk of noise-induced hearing loss (NIHL).

A literature review was conducted to determine the definition, cause, and ways to prevent NIHL in relation to the dental clinics and orthopaedic cast clinics. Also determined from a review of the literature were ways to assess and monitor the acoustics of these spaces.

Initially room acoustic measurements of background noise levels as well as reverberation times were made and frequency information on the major noise sources was obtained. This was followed by measurement of the daily noise dose exposure of staff working in the participating dental clinics and orthopaedic cast clinics.

It was found that noise dose levels did not exceed the damage risk criterion set by The New Zealand Occupational Safety and Health Service of L_{eq8h} of 85 dBA and therefore staff were considered to not be at risk of NIHL. However, the background noise levels measured may be putting healthcare workers at risk of non-auditory related effects of noise exposure, affecting work performance, cognitive abilities and vital communication between staff and patients. Healthcare workers may also be at risk of non-auditory health effects due to increased noise annoyance leading to raised stress levels, which may ultimately lead to pathophysiological changes in the myocardium. Future research in the area of noise levels in the New Zealand health industry should

be performed to obtain noise data on a larger sample and look further at the non-auditory health effects of exposure to noise in the health industry.

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NOMENCLATURE

A-weighting filter: Frequency weighting approximating the inverse of the 40 dB equal loudness curve, that is to say, the human ear's response at low to medium sound levels. It is far the most commonly applied frequency weighting and is used for all levels of sound.

Criterion level: Criterion level is the maximum averaged sound level allowed in an 8-hour period. Used for the calculation of dose.

Decibel (dB): The measurement unit for expressing the relative intensity of sound. A direct application of linear scales (in Pa) to the measurement of sound pressure leads to large and unwieldy numbers. As the ear responds logarithmically rather than linearly to stimuli, it is more practical to express acoustic parameters as ten times the logarithm of the ratio of the energy of the measured value to the energy of the reference value. This quantity has the unit of decibel or dB.

Dose: The Noise Dose is the equivalent averaged A-weighted Noise Level (taking the Threshold Level into account) using Exchange Rate = 3 dB for an 8 hour period (reference duration) relative to a maximum allowed (the Criterion Level) – expressed as a percentage.

Exchange Rate: Exchange Rate is the increase in noise level that corresponds to a doubling of the noise level. L_{Aeq} is always based on an Exchange Rate = 3 dB.

Frequency: The number of pressure variations per second. Frequency is measured in Hertz (Hz). The normal hearing for a healthy young person ranges from approximately 20 Hz to 20000 Hz (20kHz).

Frequency weighting: Our hearing is less sensitive at low and very high frequencies. In order to account for this, weighting filters can be applied when measuring sound. The most commonly weighting is 'A-weighting', which approximates the human ear's response to low – medium noise levels.

L_{Aeq} : A widely used noise parameter that calculates a constant level of noise with the same energy content as the varying acoustic noise signal being measured. The letter 'A' denotes that the A-weighting has been included and 'eq' indicates that an equivalent level has been calculated. Hence, L_{Aeq} is the A-weighted equivalent continuous noise level.

$L_{eq,d}$: The Daily Dose Exposure Level is the average A-weighted noise exposure level for a nominal 8-hour working day. Used for assessing the noise exposed to a worker during a working day.

Loudness, Loudness Level: Loudness is the subjective judgement of intensity of sound by humans. Loudness depends upon the sound pressure and frequency of the stimulus and whether the sound field is diffuse- or free-

field. The unit is the Sone. Loudness Level = $10 \cdot \log_2(\text{loudness}) + 40$. The unit is the Phone. The Zwicker method of calculation of stationary loudness based on 1/3-octave measurements is described in ISO 532-1975, Method B.

Sound: Any pressure variation that the human ear can detect. Just like dominoes, a wave motion is set off when an element sets the nearest particle of air into motion. This motion gradually spreads to adjacent air particles further away from the source. Depending on the medium, sound extends and affects a greater area (propagates) at different speeds. In air, sound propagates at a speed of approximately 340 m/s. In liquids and solids the propagation velocity is greater – 1500 m/s in water and 5000 m/s in steel.

Sound exposure level (SEL): The sound exposure expressed as a level.

Sound level or sound pressure level: The level in decibels of the pressure variation of sound.

Threshold Level: Any sound levels below the threshold level do not contribute to the Dose measurement data. For example, if you set the threshold level to 80, any sound levels below 80 dB are not taken into consideration by the instrument, when it calculates doses and time weighted averages.

Introduction

1.1 Research Outline

This section outlines the research question, importance, aims and hypothesis of the study.

1.1.1 Research Questions and Importance

Noise-induced hearing loss (NIHL) has a significant impact on the health, well-being and productivity of individuals and its cost to society. It is estimated that between 2.25% and 2.58% of the New Zealand population have NIHL or some contribution to their total hearing loss from occupational noise exposure (Laird, 2012). Current Accident Compensation Corporation (ACC) statistics indicate that the total cost exceeds \$NZ 40 million per annum with about 4000 new serious injury claims relating to NIHL being lodged every year (New Zealand Department of Labour). There has been a steady increase in claims in the recent past from 3,000 in 2001 to 5000 claims in 2008 (Thorne et al., 2008). Thorne et al., 2008, report that although recent changes to ACC funding has had an impact on the number of individuals seeking

hearing aid services, figures suggest that the number of claims for NIHL will continue to rise. It is, therefore, important to monitor noise exposure levels in the workplace to assess the need for prevention strategies and conservation programmes (Thorne et al., 2008).

Zubick et al, 1980, performed a study comparing the hearing thresholds, using pure tone air conduction audiometry, of dentists (n=137) with those of physicians (n=80). The audiometric results revealed higher hearing thresholds in dentists than those in the physician, especially at 4,000 Hz. Additionally, a significant difference in hearing thresholds was seen between the left and right ears of right-handed dentist which was not seen in their medical counterparts. The greater hearing loss in the left ear was presumed to be due to its closer proximity to the noise source (Zubick et al., 1980). Gijbels et al, 2006, used pure tone air conduction audiometry to test the hearing of 13 dental professionals and compared these results to audiograms recorded ten years earlier. The audiometric results revealed the only significant change had been in the left ear at 4,000 Hz and that hearing thresholds in the left ear was significantly higher than that of the right ear (Gijbels et al, 2006). Nevertheless, as reported in Sorainen & Rytönen, 2002, studies do exist in which no significant differences were found in hearing thresholds of dentists.

There have been similar reports of hearing loss amongst orthopaedic staff. Audiometric results obtained in a study by Willett, 1991, revealed that 11 of the 27 participants tested showed evidence of NIHL. However, Marsh et al., 2011, reported that although noise levels in an orthopaedic cast clinic fell within safety limits, staff and patients were exposure to subjectively high levels of noise, which could result in increased levels of anxiety.

To date there have been very few studies assessing noise levels in the New Zealand health industry. This study will aim to obtain some New Zealand specific data by assessing the risk of NIHL and non-auditory affects of noise in the health industry in New Zealand. The two main research questions to be answered in this study:

1. Are dental and orthopaedic staff at risk of developing NIHL?
2. Are dental and orthopaedic staff at risk of developing noise-related non-auditory health effects?

1.1.2 Aims of the Study

As previously mentioned, exposure to 8-hour continuous sound pressure levels 85 dBA and greater are known to cause NIHL. The aim of this study was to evaluate the acoustic environments and determine the spectral characteristics of major noise sources within those environments, and to obtain noise level data for New Zealand dental and orthopaedic clinics to determine whether staff in such clinics are at risk from excessive noise exposure. And hence, determine any need for prevention strategies and hearing conservation programmes.

1.2 References

- New Zealand Department of Labour. *Key facts about noise induced hearing loss*. Obtained from <http://www.dol.govt.nz/pdfs/noise-induced-hearing-loss-facts.pdf>
- Gijbels, F., Jacobs, R., Princen, K., Nackaerts, O., & Debruyne, F. (2006). Potential occupational health problems for dentists in Flanders, Belgium. *Clinical Oral Investigation*, 10, 8-16.
- Laird, I. (2012). *The epidemiology and prevention of NIHL in New Zealand*. Paper presented at the Symposium on Health and the Environment at Work - the Need for Solutions, Wellington, NZ.
- Marsh, J. P., Jellicoe, P., Black, B., Monson, R. C., & Clark, T. A. (2011). Noise levels in adult and pediatric cast clinics. *The American Journal of Orthopedics*, 40(7), E122-E124.
- Sorainen, E., & Rytönen, E. (2002). Noise level and ultrasound spectra during burring. *Clinical Oral Investigation*, 6, 133-136.
- Thorne, P. R., Ameratunga, S. N., Stewart, J., Reid, N., Williams, W., Purdy, S. C., Dodd, G., Wallaart, J. (2008). Epidemiology of noise-induced hearing loss in New Zealand. *The New Zealand Medical Journal*, 121(1280), 1-9.
- Willett, K. M. (1991). Noise-induced hearing loss in orthopaedic staff. *The Journal of Bone and Joint Surgery*, 73 B(1), 113-115.
- Zubick, H. H., Tolentino, A. T., & Boffa, J. (1980). Hearing loss and the highspeed dental drill. *American Journal of Public Health*, 70(6), 633-635.

2

The Literature Review

2.1 The Literature Review

This section identifies and reviews previous work on the definition, cause, and prevention of noise-induced hearing loss (NIHL) in relation to the dental surgeries and orthopaedic cast clinics and considers ways to assess and monitor the acoustics of these spaces.

2.1.1 Sound, Noise and Hearing

In everyday life we are surrounded by sound. Sound has many functions; some sounds may be perceived as being enjoyable such as music or bird song, while other sounds may act as a warning signal such as the sound of a car horn, sound is also a vital component of communication. Often however, sound may be unpleasant, annoying and unwanted, these sounds are referred to as noise.

2.1.1.1 Sound

Sound may be defined in terms of either a psychological or a physical dimension (Yost & Neilson, 1997). From a psychological perspective sound is a sensation perceived by the ear and is defined as pressure waves that travel through a medium carrying information, signal or communication. On the other hand, ‘noise’ is also a sensation perceived by the ear but is defined as unwanted sound, carrying no useful information. The psychological definitions of sound and noise include such aspects as pitch, loudness and timbre (Speaks, 2005). Whether the sensation is perceived as sound or noise not only depends on these aspects of sound quality but also on those perceiving the sound. The sound of a twin turbo engine of a new car may be music to the ear of its owner but may be annoying to a neighbour studying for an exam.

From a physical perspective, sound is produced when an object with the properties of inertia and elasticity is forced into vibration. Waves of particle compression and expansion within the object cause small pressure variations, which propagate as a longitudinal wave through a media, most commonly air, resulting in an “audible” sound (Stach, 1998). Whereas the psychologist would refer to the attributes of pitch, loudness and timbre, the physicist refers to the parameters of frequency, sound pressure and tonal characteristics. To the physicist, sound and noise are analogous (Yost & Neilson, 1997; Yost, 2000; Speaks, 2005).

2.1.1.2 Noise Exposure

It is the physicist’s definition of noise that is relevant to NIHL, as any

sound can contribute to the disorder regardless of its source or whether it is perceived as desirable or not. In terms of hearing loss, mechanical noise, music, machinery and speech are all potentially as risky as each other. The sound pressure level, duration and cumulative exposure to a sound determine its pathological impact upon the ear. As noise is a form of energy, noise exposure is a combination of both the sound pressure level (SPL) and the duration of the noise. For example, exposure to a loud sound for one hour is less harmful than exposure to the same sound for four hours. Therefore in order to determine the risk in terms of hearing thresholds posed by a particular sound environment both the sound pressure level and the duration of the exposure must be measured (Royster, Royster, & Killion, 1991).

There is awareness in most industrialized countries of the need to protect workers against the risk of hearing loss due to hazardous noise levels in workplace environments. Acceptable occupational noise levels differ throughout the world. A list of recommended maximum noise levels, for a given exposure period in industrial environments, was adopted by the American Occupational Safety and Health Administration (OSHA) in 1971. OSHA allows for a maximum permissible exposure limit at 90 dB with a 5 dB exchange rate, which is measured as a time-weighted average exposure level (TWA). The Workers' Compensation Boards of Canada have also adopted these recommendations. These levels, however, are approximately five decibels above those recommended by the American Otological Association (Lipscomb, 1994). The New Zealand Occupational Safety and Health Service (2003) has set a "safe level" of continuous noise exposure at no more than 85 dBA (i.e., decibels measured on the A scale of a sound level meter) based on

an 8 hour daily, 40 hour week work period, with a 3dB exchange rate (OSH, 2002).

To allow for exposure durations other than eight hours, an exchange rate based on the “equal energy principle” is used to determine the permissible exposure time. Regardless of the temporal pattern of the noise, equal amounts of acoustical energy are considered to be equally hazardous (Henderson, Subramanian, & Boettcher, 1993). The New Zealand Occupational Safety and Health Service has set a damage risk criterion, the recommended noise level for a given exposure period, of 85 dBA with the exchange rate at 3 dB. That is, there is identical risk to hearing thresholds for every 3 dB increase in sound pressure level when there is a corresponding halving of the duration of exposure (OSH, 2002).

2.1.1.3 Noise-Induced Hearing Loss

Noise is one of the most pervasive occupational hazards found in a wide range of industries, causing NIHL to become one of the most prevalent occupational health disorders worldwide (Kircher, 2003; Haller & Monygomery, 2004; Kircher et al., 2012). Exposure to high sound pressure levels (SPL) causes auditory fatigue resulting in damage to the hair cells of the cochlea and a shift in hearing thresholds. It is a preventable hearing disorder that affects people of all ages and demographics (Henderson et al., 1993; Haller & Monygomery, 2004)

NIHL is sensorineural hearing loss (SNHL) that results from intermittent or continuous exposure to hazardous levels of noise. NIHL generally affects both ears equally and develops slowly over a number of years.

An ear, nose and throat surgeon (ENT) makes the diagnosis of NIHL after careful consideration of the worker's industrial and recreational noise exposure history, along with other factors that may affect auditory thresholds. Other causes of SNHL include a wide variety of genetic disorders, infectious diseases, pharmacological agents, head trauma, therapeutic radiation exposure, neurologic disorders, cerebral vascular disorders, immune disorders, bone disorders, central nervous system neoplasms, and the aging process. A full medical history can help in determining whether any of these conditions could contribute to an individual's hearing loss (Kircher, 2003; Kircher et al., 2012).

2.1.1.4 Characteristics of Noise Induced Hearing Loss

NIHL affects the hair cells of the cochlear typically in both ears, as noise exposure is generally symmetrical. There may be a unilateral NIHL in the case of firearm use (Kircher et al., 2012).

The audiogram typically shows a “notched” configuration between 3000 Hz and 6000 Hz with recovery at 8000 Hz. The notch results from amplification of the acoustical energy of high frequency sounds due to the resonant characteristics of the ear canal (WHO, 1997; Venema, 2006) and is dependent on the frequency of the damaging noise (Kircher, 2003; Kircher et al., 2012). With continued noise exposure, adjacent frequencies are affected making speech recognition difficult especially if combined with effects of age related hearing loss, presbycusis.

The effects of NIHL and presbycusis are cumulative and are a major cause of handicap in the elderly (WHO, 1997). The maximum hearing loss due

to noise exposure is 40 dB at low frequencies and 75 dB at high frequencies but when the effects of presbycusis are added the thresholds may become greater (Kircher, 2003; Kircher et al., 2012).

2.1.1.5 Tinnitus

Tinnitus is the false sensation or perception of sound in the head in the absence of an acoustic signal (Stach, 2003) and although common it is poorly understood. It is frequently associated with hearing loss but has also been reported in people who have hearing thresholds within normal limits (Lookwood, Salvi, & Burkard, 2002).

Tinnitus is most commonly experienced as a ringing sound in the head which may be transient, lasting a few seconds or may be permanently perceived (Lookwood et al., 2002). A permanent tinnitus may be extremely distressing for some individuals, adversely affecting their quality of life. Although many individuals can ignore the tinnitus others are troubled by sleep disturbances, increased levels of annoyance and anxiety, and depression resulting in difficulty concentrating and decreased productivity (Carmen, 1999).

In a small percentage of cases tinnitus points to the presence of underlying pathology such as a tumour however the majority of cases are of unknown aetiology. Tinnitus is frequently associated with noise trauma, the exact incidence is difficult to determine (Lookwood et al., 2002) but is reported to be between 50% and 90% of cases (World Health Organization, 2011).

2.1.1.6 Classification of NIHL

NIHL generally occurs slowly over time and the full effects are usually not realized until after 10-15 years of chronic noise exposure (Miller, 1974; Albera, Lacilla, Piumetto, & Canale, 2009) however, some NIHL may be evident after a single exposure to loud noise (Melnick, 1991). Noise related hearing changes can be categorized into three groups: acute acoustic trauma, noise-induced temporary threshold shift (TTS), and noise-induced permanent threshold shift (PTS).

Acute acoustic trauma refers to a sudden permanent hearing loss which results from a single exposure to a sudden burst of intense impulse sound, such as an explosive blast or gun shot (Henderson, Subramanian, & Boettcher, 1993). Exposure to impulse sound results in mechanical damage to the sensory hair cells of the cochlea causing an instantaneous permanent SNHL.

Noise-induced TTS refers to a reduction in hearing sensitivity due to exposure to loud noise in which there is a full recovery to pre-exposure hearing thresholds. Hearing thresholds may take minutes or may take up to a couple of days to fully recover to pre-exposure levels after cessation of the noise (Kircher, 2003; Feuerstein & Marshall, 2009; Kircher et al., 2012). The reduction in hearing sensitivity may be accompanied by a possible subjective feeling of aural fullness due to the reduction in high frequency sensitivity, and tinnitus.

The severity of the noise-induced TTS is correlated to the sound pressure level and duration of the noise exposure. Exposure to higher levels of

noise will result in a more severe TTS while the shift grows during the first eight hours of noise exposure then plateaus (Feuerstein & Marshall, 2009).

Noise-induced PTS refers to a reduction in hearing sensitivity due to exposure to loud noise in which hearing thresholds fail to recover to pre-exposure levels. A PTS usually develops slowly over a number of years and emerges when there is insufficient recovery from TTS due to repeated noise exposure (Albera et al., 2009).

2.1.2 Confounding Factors in NIHL

NIHL is rarely the sole cause of a sensorineural hearing loss. Other factors that may contribute to raised auditory thresholds include presbycusis and socioculus as well as individual susceptibility.

2.1.2.1.Presbycusis and Socioculus

Noise exposure, confounded by ageing, explains the variance of the hearing loss in 40% of NIHL (Pyykko, Starck, Toppila, & Kaksonen, 1998). The audiometric profile of NIHL has a notch at 3 kHz, 4 KHz or 6 kHz while the audiometric changes due to age (presbycusis) show a high frequency threshold shift vary according to the associated pathology. The hearing losses due to presbycusis and NIHL are additive (WHO, 1997).

Presbycusis is the gradual loss of hearing sensitivity and acuity that is solely due to ageing. The physiological age-related changes include the slowing down of reproduction of some cells while others show an increased rate of production. This leads to decreased auditory function, intracellular and

extracellular deposition of various materials, such as cholesterol, causing neural degeneration, and changes in the structural characteristics of support structures in the auditory system (Ward, 1971; Parham, Gates, Dobie, McKinnon, & Backous, 2010).

Presbycusis by definition is due to ageing alone and therefore should not include the effects of exposure to noise above 80 dBA, ototoxic drugs and chemicals, head trauma, barotrauma, middle ear infection or a genetic predisposition to hearing loss. Neither should it include the effects of other age-related systemic diseases such as cardiovascular disease, diabetes and osteoporosis that also impact on auditory function (Ward, 1971; Parham et al., 2010). It is however very difficult to tease out the effects on the auditory system caused by the aging process alone, audiometric measurements would need to be performed on normal healthy subjects raised in a germ and noise free environment over many years.

In 1961, Dr. A Glorig, founder of the American Auditory Society, and forensic and industrial ear specialist, coined the term sociocosis. Sociocosis refers to the loss of hearing sensitivity and acuity associated with the exposure to the auditory hazards of everyday life and excluding the effects of occupational noise exposure and presbycusis (Glorig & Nixon, 1962; Ward, 1971). It includes hearing damage due to middle ear pathology from infections, barotrauma, conductive losses, in addition to the sensorineural losses produced by recreational noise exposure, exposure to ototoxic substances, and diseases such as mumps, measles and meningitis. It is impossible to determine the effect of sociocosis on an individual's hearing as the variables are so great (Ward, 1971).

Non-occupational/recreational noise exposure from a variety of sources, such as loud music, weapons firing, motor sports, etc (Kircher et al., 2012). Voluntary exposure to noise at concerts, nightclubs and the use of personal listening devices (PLD), such as MP3 players, along with other everyday leisure activities has the potential to cause hearing damage. Estimating hearing risk due to recreational exposure is difficult because of its intermittent and irregular nature. However, when tested, PLDs have been found to have a maximum output of 96 to 107 dB depending on the make and model, and the transducer used, that is, speakers, headphones or ear-buds etc. Using noise criteria of 85 dB with an exchange rate of 3 dB with an output of 100 dB, a listener should limit exposure to a maximum of 15 minutes at maximum levels (Williams & Purnell, 2010; Carter, Gilliver, Macoun, Rosen, & Williams, 2012). Although most PLD users would not use the device at maximum levels, measurements of actual listening levels and self reported durations suggest 17-25% of PLD users listen at potentially harmful levels (Carter et al., 2012).

2.1.2.2 Individual Susceptibility

As previously discussed, NIHL may result from exposure to noise and depends on the sound pressure level and the duration of exposure. Exposure to sound levels of 75 dBA and below is considered to be harmless, whereas those 85 dBA and above may result in permanent hearing loss. However the degree of risk to an individual is also dependent on a number of other factors such as individual age, susceptibility and comorbidity (Setcos & Mahayuddin, 1998; Bhat, Jyothi, Kadanakuppe, & Ramegowda, 2011).

Not everyone who is exposed to noise levels greater than 85 dB for 40 hours a week over their lifetime will experience a NIHL. Studies have shown that a broad range of individual sensitivity to noise exposure (Prasher, 1998; Pyykko et al., 1998) as can be seen in Table 2.1.

Level of exposure in dBA Leq (eight hours)	Ten-year exposure Number of persons per hundred	Lifetime exposure Number of persons per hundred
100	17	32
90	5	11
80	1	3

Table 2.1. Percentage of Individuals Likely to Suffer a 50 dB Hearing Loss: dB(A) means an A-weighted filter was used to measure the sound level; Leq (eight hours) means the equivalent continuous sound level normalized at eight hours (Prasher, 1998)

Several biological and environmental factors have been proposed to explain the differences in NIHL among individuals and why not all individuals exposed are affected (Prasher, 1998). Factors such as elevated blood pressure, altered lipid metabolism, the presence of vibration white finger (VWF), genetic factors and an individuals use of drugs, both therapeutic and recreational, and alcohol and tobacco habits are believed to contribute to NIHL (Pyykko et al., 1998; Starck, 1998).

Some studies have found a correlation between elevated blood pressure and NIHL. However, it is thought that elevated arterial blood pressure may accelerate age-related hearing loss confounding the effects of NIHL (Pyykko et al., 1998).

Skin pigmentation is thought to have an effect on the vulnerability to NIHL. Animal and human studies have shown those with dark skin have reduced threshold shifts when compared to those with skin and blue eyes (Prasher, 1998). Higher levels of melanocytes are thought to have protective capabilities in the inner ear against damage caused by noise exposure (Pyykko et al., 1998).

A gender difference in susceptibility to NIHL has been reported, with males being more susceptible than females (Damen, Pennings, Snik, & Mylanus, 2006). The difference was thought to result from disparities in recreational noise exposure (Pyykko et al., 1998). Gender differences are also present in age-related hearing loss with males showing higher thresholds than women (Hood, 1998).

Ototoxic drugs and other chemicals appear to exacerbate the damaging effects of noise exposure. Serum magnesium levels have been shown to reduce susceptibility to the risk of NIHL in a given noise exposure. Magnesium deficiency results in vasoconstriction and reduced cochlear blood flow thereby increasing susceptibility to noise-induced damage while magnesium supplementation offers protection against threshold shifts (Attias, Bresloff, Joachims, & Ising, 1998).

Although there are insufficient data available on the relationship between NIHL and genetic background there are indications that genetic factors play a significant role in the development of age-dependent hearing loss and NIHL. Genetic hearing loss is divided into hereditary or sporadic gene transformations. Syndromic hearing loss is part of a collection of specific signs and symptoms associated with a syndrome. A non-syndromic hearing loss is not associated with other signs and symptoms and is often difficult to separate from NIHL. Connexin 26 (Cx26) is the most common of the 33 localized loci for non-syndromic hearing loss and is found in 3% of the population (Pyykko et al., 1998). Animal studies using inbred mice have also demonstrated genetic susceptibility to NIHL (Prasher, 1998).

2.1.3 Non-auditory Health Effects of Noise Exposure

As well as the audiological effects of noise exposure, variations in heart rate, blood pressure, respiration, blood glucose and lipid levels, psychological consequences such as annoyance, mental fatigue and a reduction in efficiency may also contribute (Bhat et al., 2011). There is evidence for underlying causal connections between noise and various health effects. Increased levels of catecholamine and cortisol associated with stress and anxiety results in elevated blood pressure, increased heart rate and compromised immunity, stress influences plasma cholesterol, which is probably involved in cardiovascular disease. Another important example of possible mechanism for health effects of noise is uncontrollability and learned “helplessness” effects (Job, 1996).

2.1.3.1 Noise Annoyance

Although annoyance is a common and well documented subjective response to noise it is probably the most challenging to describe (Fidell, 1979). Annoying noise has been described as a sound that would cause an individual or group of individuals to reduce or avoid the noise or to leave a noisy area (Molino, 1979). Annoyance to a given sound varies widely amongst individuals. Not only is the level of annoyance dependent on sound pressure level, duration and tonal characteristics, but it is also a function of individual sensitivity and attitude towards the noise along with the degree of activity disruption caused (Stansfeld & Matheson, 2003).

A number of studies looking at the community effect of aircraft and traffic noise have found a dose-response relationship between noise intensity

and levels of annoyance (Stansfeld & Matheson, 2003). That is, louder noises are generally considered to be more annoying than quieter noises. The presence of tonal components influences the degree of annoyance as does the number of tonal components, that is, noise with multiple tonal components is more annoying than noise with a single tonal component (Landstrom & Akerlund, 1995). Noise with higher tonal characteristics is perceived as more annoying than noise with lower tonal characteristics. Other secondary acoustical features of noise that affect the degree of annoyance felt include spectral complexity, frequency and/or sound pressure level fluctuations, localization of the noise source and the rise-time of the noise (Molino, 1979).

Noise intensity accounts for only 25% of the variance in levels of annoyance while such factors as personal attitudes and beliefs about the noise account for about 50% of the variance (Smith, 1991). Noise is seemingly more annoying if it is perceived to be unnecessary or if those responsible for the noise are thought to be indifferent toward the welfare of those exposed to the noise. Annoyance is greater when the exposed person has no control over the noise, when noise is intrusive, associated with fear or believed to be harmful to the health of the individual (Molino, 1979; Smith, 1991; Stansfeld & Matheson, 2003). Noise annoyance is greatest when noise is present at night time or in the early hours of the morning (Raney & Cawthorn, 1979; Stansfeld & Matheson, 2003).

Noise sensitivity is considered a stable personal trait. Individual sensitivity to noise means that exposure to noise results in different levels of annoyance amongst individuals. Shepard, Welch, Dirks & Mathews (2010) suggested that “noise sensitivity has no relationship to auditory acuity, instead reflected a judgmental, evaluative predisposition towards the perception of

noise” and concluded that “noise sensitivity can degrade quality of life through annoyance and sleep disruption” (Shepard, Welch, Dirks, & Mathews, 2010).

2.1.3.2 Noise effects on work performance

Noise levels in work environments have been shown to affect work efficiency and performance. Behavioural responses to noise are usually explained in terms of arousal theory which states “there is an optimum level of arousal for efficient performance; below this level behaviour is sluggish and above it, tense and jittery” (Bies & Hansen, 1988; Hansen, 2005). That is, with increased noise levels the efficiency and performance of complex, multifaceted tasks decreases. On the other hand, an increased noise level may lead to an increased productivity of simple, repetitive or monotonous tasks (Bies & Hansen, 1988; Suter & Berger, 2002; Hansen, 2005). Tasks involving sensory input are particularly susceptible to increases in noise levels (Broadbent, 1979; Suter & Berger, 2002).

When noise levels exceed those required for optimal arousal, workers become less efficient and irritable. Increased noise levels also correlate with Increased incidences of accidents (Broadbent, 1979; van Dijk, 1990), antisocial behaviour and disciplinary actions (van Dijk, 1990), and decreased cooperation amongst colleagues (Suter & Berger, 2002). Broadbent (1979) reports that the frequency content of the noise also has an effect on productivity with high frequency noise resulting in increased irritability and decreased productivity.

Evidence suggests that cognitive functions involving central processing and language comprehension and concentration are affected by chronic exposure to noise (Stansfeld & Matheson, 2003).

2.1.3.3 Speech Intelligibility

Excessive noise in the workplace that masks warning signals can have a detrimental affect on worker safety; it can also impact on a worker's ability to understand spoken communication. Normal conversational speech is in the range of 55 to 65 dBA. For speech to be intelligible it must be heard at the listener's ear at sound pressure levels greater than that of any background noise (Webster, 1979). Therefore any noise within this range or louder can mask speech and reduce intelligibility in face-to-face conversation, telephone conversations and other more sophisticated means of communication (Suter & Berger, 2002). It is important to note that people with otherwise unnoticeable hearing loss find it difficult to understand spoken words in noisy surroundings.

The overall sound pressure level and frequency content of speech varies over the course of conversation. As the level of background noise increases more vocal effort is required from the speaker to maintain the signal to noise ratio; speech intelligibility, however, is detrimentally affected by this added effort. The extra vocal effort required may result in hoarseness, vocal nodules and other vocal cord pathology (Smith, 1991; Suter & Berger, 2002). As well as the stress placed on speaker through extra vocal effort, the listener must strain to hear and understand the spoken message (Suter & Berger, 2002).

The interplay of various factors need to be taken into consideration when dealing with noise in the work place, such as the distance between speaker and listener, background noise levels, room acoustics and the importance of the message being conveyed. Ambient noise level recommendations and predicted communication difficulties in workplace environments have been developed. Table 2.2 shows the average sound pressure levels required for different levels of vocal effort at a distance of 1 metre under quiet conditions.

Vocal Effort	A-weighted Sound Level (dBA)
Maximum	88
Shout	82
Very loud	74
Raised	65
Normal	57
Relaxed	50
Whisper	40

Table 2.2. Vocal Effort vs. A-weighted Sound Levels. A-weighted sound levels (long-term averages) for different vocal efforts under quiet conditions, at 1m (Webster, 1979)

Figure 2.1 shows the relationship between the A-weighted sound level of background noise and the permissible distance between listeners and the talker for “satisfactory communication,” with at least 95% of the sentence understood correctly. From this data it can be seen that satisfactory communication is achievable with normal vocal exertion when the speaker is at a distance of 5 m from a noise having an A-weighted sound level up to 50 dB(A). For every 10 dB increase in noise levels above 50 dB(A) the speaker is required to raise their voice level by 3-6 dB so as to be clearly understood. Figure 2.1 applies to situations where speech reaches the ears of a listener without reflections from interior surfaces of a room. Reverberant sound decreases speech intelligibility.

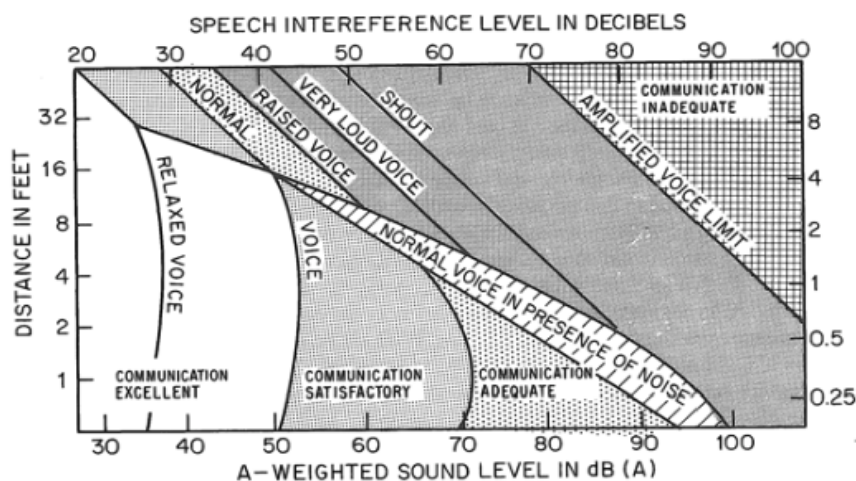


Figure 2.1 Speech Interference Graph Acceptable distance between speaker and listener for various vocal effort when not facing each other. Distance is plotted as a function of the A-weighted sound level (lower horizontal axis) and speech interference level (upper horizontal axis). A 5 dB background noise level is acceptable if the speaker and listener are facing each other (Webster, 1979).

The maximum tolerable background noise level for adequate speech intelligibility is specified in relation to the intended purpose of the space. The recommended maximum background noise level for professional rooms such as dental clinics, surgeries and consultation rooms is 40-45 dB(A) with recommended reverberation times of between 0.4 seconds and 0.7 seconds (AS/NZS 2107, 2000). The A-weighted level simulates the response of the ear at low levels, and has been found to correlate well with subjective response to noise (Harris, 1979; Bies & Hansen, 1988; Hansen, 2005).

Although, with difficulty, conversation is possible at a distance of one metre for a short time in the presence of noise as high as 78 dBA, for prolonged conversations, the background noise level must be lower than 78 dBA. It is recommended that the A-weighted sound level, in work spaces where speech communication is essential, not exceed 62 dBA; this level permits satisfactory communication at a distance of 2m (Webster, 1979). Webster (1979) formulated the following table as an indication of the effects on vocal effort in background noise.

Communication	Below 50dB(A)	50-70 dB(A)	70-90 dB(A)	90-100 dB(A)	110-130 dB(A)
Face-to-face (Unamplified speech)	Normal voice at distances up to 6 m	Raised voice level at distances up to 2m	Very loud or shouted voice level at distances up to 50cm	Maximum voice level at distances up to 25cm	Very difficult or impossible, even at a distance of 1cm
Telephone	Good	Satisfactory to slightly difficult	Difficult to unsatisfactory	Use-press-to talk switch and an acoustically treated booth	Use special equipment
Intercom system	Good	Satisfactory	Unsatisfactory using loudspeaker	Impossible using loudspeaker	Impossible using loudspeaker
Type of earphone to supplement loudspeaker	None	Any	Use any earphone	Use any in muff or helmet except bone conduction type	Use insert type or over-ear earphones in the helmet or in muffs; good at 120 dB(A) on short term basis
Public address system	Good	Satisfactory	Satisfactory to difficult	Difficult	Very difficult
Type of microphone required	Any	Any	Any	Any noise cancelling microphone	Good noise cancelling microphone

Table 2.3: Speech Communication Capability vs. Background Noise Level. Affect of background noise, in dBA, on various forms of speech communication (Webster, 1979)

2.1.3.4 Sleep disturbances

Sleep is essential to health and wellbeing, providing a period of rest and preventing fatigue. Functions of sleep range include growth and restoration of the immune, nervous, muscular and skeletal systems and is plays a vital role in memory consolidation. Most of the studies on sleep disturbances have looked at the effects of nighttime environmental noise especially aircraft noise and traffic noise either by self-assessment questionnaires or in a laboratory setting. Researchers have measured the effects of sleep disturbance looking at the number and duration of nocturnal disturbances and the resulting changes in “sleep architecture,” that is the quality of sleep, the organization of sleep stages and body movements, and number of arousals (Kryter, 1972).

Sleep studies show that noise not only has an immediate effect on the quality of sleep but also can have long-term effects on mental and physical

health. The World Health Organization (WHO, Night Noise Guidelines for Europe) has set an average nighttime noise exposure limit of 30 dBA. This level corresponds to the sound from a quiet street in a residential area. Nighttime noise acts as a stressor on the body and can initiate an autonomic response with increases in blood cortisol, adrenaline and noradrenaline levels (Kryter, 1972). Exposure to levels of night time noise greater than 30 dBA are reported to result in sleep disturbances and insomnia. The flow on effect from these disturbances includes increased fatigue and decreased performance along with a possible negative effect on temperament. When nighttime noise levels exceed 55 dBA the resulting stress on the body and the subsequent raised cortisol, adrenaline and noradrenaline levels are associated with long-term health effects on the cardiovascular system (WHO, 1999).

Industrial noise does not have a direct affect on sleep. It does however act as a stressor on the body producing an autonomic response. The resulting increase in serum cortisol, adrenaline and noradrenaline levels trigger the same physical and psychological changes in the body as seen in environmental noise/sleep disturbances studies (Kryter, 1972).

2.1.3.5 Cardiovascular Effects

Noise levels below the noise damage criterion of 85 dBA although considered to have no significant effect on the auditory system do however have adverse non-auditory effects. Noise below 85 dBA as well as causing annoyance, sleep disturbances and cognitive impairment, has the potential to trigger the release of stress hormones such as catecholamines (adrenaline and noradrenaline) and cortisol (Ising, Babisch, & Kruppa, 1999; Spreng, 2000;

Babisch, 2003, 2011). The increased concentrations of these hormones in the blood trigger the “fight or flight” response in the body (Babisch, 2003). These stress hormones are associated with the accelerated ageing of heart muscle (the myocardium) and therefore increases the risk of developing ischaemic heart disease and myocardial infarction (Ising et al., 1999; Willich, Wegscheider, Stallmann, & Keil, 2006).

Subjects experimentally exposed to aircraft noise with maximal levels of 55-65 dBA are found to have increased levels of cortisol (Spreng, 2000). Increases in cortisol levels have been observed in subjects attempting to perform complex mental tasks, including arithmetic calculations and decision making, in the presence of noise even at low levels (Babisch, 2003). Ising, et al (1999) reported increased levels of noradrenaline and cortisol in persons exposed to acute and habitual work noise. Increased adrenaline release is associated with the perception of noise causing discomfort and emotional distress and with unpredictable impulse noise (Babisch, 2003).

Research looking into general stress and noise stress has shown that although long-term noise exposure may lead to habituation and a reduction in acute stress effects long-term exposure may nevertheless result in physiological damage (Babisch, 2003). Long-term exposure leads to an acute increased in cortisol excretion, which is followed by a normalization period of about two weeks and a subsequent long-term increase of cortisol levels (Ising et al., 1999). However, intermittent industrial noise has shown greater increases in serum noradrenaline levels than when subjects are exposed to steady state noise (Babisch, 2003).

The results from studies looking at the noise-induced increase in stress hormones in both animals and humans have been found to be qualitatively

similar. This means that the long-term health effects of noise-induced stress can be studied qualitatively in the animal model (Ising et al., 1999). Initial studies on noise stress concentrated on noise-induced vasoconstriction and increases in blood pressure, however epidemiological evidence points more to an increased risk of myocardial infarction and ischaemic heart disease than hypertension (Ising et al., 1999; Willich et al., 2006).

Animal studies have revealed a chronic increase of noradrenaline with persistent repeated noise exposure. Moderate chronic noise exposure has been found to increase the ratio of calcium to magnesium (Ca/Mg) in the myocardium and vascular walls resulting in biological aging and a decreased life expectancy. Ca/Mg shifts of this nature have been found on post-mortem examination of heart tissue from ischaemic heart disease sufferers and are also associated with the normal ageing process (Ising et al., 1999).

2.1.4 Room Acoustics in the Workplace

Uncomfortable noise levels in the workplace can affect workers psychologically, sociologically and physically, which has been shown to affect concentration levels, decrease productivity and increase absenteeism (Kua, Lee, & Mahbub, 2010).

2.1.4.1 Noise in the Workplace

Noise problems comprise three components, the *source*, the *path*, and the *receiver*. The sound level within a room or building is affected by interplay of the building's location, that is, a quiet or noisy setting, and its interior, structural, and mechanical systems. The exact amalgamation of these factors is dependent on the proposed use of the building. It is therefore important

that due consideration is given to all of these factors throughout the planning, designing and construction processes. (Kua et al., 2010).

Even before construction begins, the involvement of architects, engineers, building technologists, and constructors is important in the development of the building's acoustical characteristics. What the buildings is to used for, how the space is to be divided up, what materials and structural elements to be used needs to be considered (Iannace, Lembo, Maffei, & Nataletti, 2006). These factors determine the acoustic environment within the space and how the sound transmitted from adjacent spaces will interact (Gastmeier & Aitken, 1999).

The materials of the wall, floor and ceiling materials, and the adjacent spaces determine the amount of sound transmitted through to adjacent spaces. The absorbency or reflective nature of surface linings has an affect on both the noise level and the nature of the sound within a space.

2.1.4.2 Reverberation Time

When a noise is produced in an enclosed space multiple reflections are generated. This reflected sound results in a build up in the total sound level. Once the original noise is discontinued, the reflections decrease and the total sound level decays over a period of time. The time it takes for the sound level to decay is called the reverberation time (Sharland, 1972).

The reverberation time of a room is influenced by the size and shape of the room and its features and by the absorbency of the surface materials in the room (Schroeder, 1980). The reverberation time (RT60), the time taken for the total sound pressure level to decay by 60 dB, is used to quantify the acoustic environment of a room (Gastmeier & Aitken, 1999). Reverberation

time is frequency dependent, however the RT60 values are generally recorded at a mid range level (500 Hz and/or 1000Hz), the centre of the frequency range crucial to speech intelligibility. While the optimal range for RT60 for symphonic music appreciation is 1.6 -2.4 seconds (s), an RT60 of around 1.5s is required for good speech intelligibility (Gastmeier & Aitken, 1999). Speech intelligibility reduces as the RT60 increases.

As already mentioned the RT60 is the time taken for a sound to decay by 60 dB once the sound source has been removed. In many environments the ambient noise is too high to be able to generate the extra 60 dB to be able to measure the RT60. Noise within a confined space is known to decay linearly (Bies & Hansen, 1988), therefore, it is possible to extrapolate the RT 60 from the RT20 or RT30 measurements where RT20 and RT30 are the time required for the noise level to drop by 60 dB extrapolated from the decay rate of the noise level measured over 20 or 30 dB of decay respectively.

In ideal situations the normal-hearing listener can automatically and effortlessly process speech signals. However, when the speech signal is degraded in the presence of competing background noise and reverberation, a lot more effort is required (Feston, George, Goverts, & Hougast, 2010). The reverberation time in an enclosed space can be reduced by either making the space smaller, which is not always possible, or by altering the absorbency characteristics of the space (Gastmeier & Aitken, 1999). Increasing the absorbency of the surface linings of the space will result in a shorter reverberation time, a decreased noise level and a less degraded speech signal. The recommended sound levels and reverberation times for building interiors is set out in the Australian Standard/New Zealand Standard AS/NZS 2107:2000.

2.1.4.3 Noise Dosimeter

The noise dosimeter is a small, specialized sound level meter (SLM) designed to measure an individual's exposure to noise. The dosimeter is small enough to be worn on a worker's belt or shirt pocket with a small microphone positioned at ear level. Dosimeters are frequently used in industrial environments to monitor an individual worker's noise exposure and automatically calculate the noise "dose" integrated over a period of 8 hours (Peterson, 1979).

A noise dose is the amount of sound received by a worker expressed as a percentage of an eight-hour daily allowable dose for a forty-hour working week. What constitutes a daily dose is not universal. The American Occupational Health and Safety Administration (OSHA) use a 90 dB noise criterion with a 5 dB exchange rate however, most authorities worldwide, including New Zealand, use an 85 dB(A) noise criterion with a 3 dB exchange rate whereby an increase of 3 dB in sound pressure level halves the permissible exposure period (OSH, 2002).

2.1.4.4 The Lombard Effect

The Lombard effect is an involuntary reflexive vocal response by speakers to the presence of background noise, that is, with an increase in background noise a person will naturally elevate their level of vocal effort (Patel & Schell, 2008).

The Lombard effect is thought to work at a neural level in sets of audio-

vocal neurons in the peri-olivary region and the pontine reticular formation. Although the Lombard effect is reflexive, higher cortical areas of the brain are used to modulate vocal effort with respect to social context (Zollinger & Brumm, 2011).

There are a number of other vocal adjustments associated with the Lombard effect, such as, a raised fundamental frequency, flattened spectral envelope and elongated duration of speech sounds, which are collectively referred to as “Lombard speech”. The voice parameters of Lombard speech differ from those of voluntary loud speech, where the speaker only raises the volume of their voice (Zollinger & Brumm, 2011).

The Lombard effect is relevant in architectural acoustics and design where consideration must be given to ways in which unwanted noise could be reduced and speech intelligibility enhanced.

2.1.5 Noise Levels in the Health industry

This section reviews previous work on NIHL in relation to the fields of dentistry and orthopaedics.

2.1.5.1 Dental Professionals and Hearing Loss

There is a growing body of evidence suggesting that dental professionals are exposed to a number of occupational health risks on a daily basis. The list includes musculoskeletal problems, neurovascular disorders, vision complaints, infections, allergies, psychological stress, kidney disease and disturbances in short-term memory (Gijbels et al., 2006). Although there

is some debate, there is evidence to suggest that dental professionals are also at risk of NIHL (Gijbels, Jacobs, Princen, Nackaerts, & Debruyne, 2006; Mervine, 2007).

Numerous studies examining noise levels and their effects on dental professionals were carried out in the 1960s showing the existence of a minimal, high frequency sensorineural hearing loss. In 1988 a study of 68 dentists with 25 years or more experience showed higher than expected thresholds at 4, 6 and 8 kHz, however as reported by Sorainen and Rytönen, other studies published during the same period reported no significant differences between dental practitioners and the general public (Sorainen & Rytönen, 2002) or that sound levels were too low to cause damage (Gijbels et al., 2006).

Zubick et al. (1980), in a study of 137 dentists, found higher hearing thresholds, especially at 4000 Hz, than a control group of physicians (n=80). The pattern of hearing loss was consistent with that of noise trauma, showing a “noise notch” at 4000 Hz and recovery at higher frequencies. Although the hearing losses were only considered to be mild, the clinicians involved were experiencing some communication difficulties (Zubick et al., 1980).

Zubick et al (1980) furthermore reported that hearing thresholds in the left ear were elevated in right-handed dentists, which he presumed correlated with the left ear’s proximity to the noise source. This difference was not seen in members of the control group. Gijbels et al. (2006), in a study of right-handed dentists (n=13) in Belgium, also reported elevated hearing thresholds at 4000 Hz with a small but significantly greater hearing loss in the left ear at 250 and 4000 Hz.

One tool frequently used to gather information on aural health amongst dental practitioners is questionnaire-based surveys. Three such surveys showed self-reported hearing problems amongst dentist of 5% from the United Arab Emirates and 11.3% of dentists from Thailand (Messano & Petti, 2012), in the Belgium study 19.6% of dentists reported auditory disorders, which showed a significant correlation with age (Gijbels et al., 2006). Messano and Pettis' own study revealed that dentists were twice as likely to report presumptive hearing loss than their medical practitioner counterparts. Most questionnaire studies were based on perceived symptoms only as no audiometric data was obtained.

Dental professionals are exposed to equipment that emits differing levels of noise. Dental equipment such as high-speed handpieces and ultrasonic scalers being identified as the major noise sources (Sorainen & Rytönen, 2002; Fernandes, Carvalho, Gallas, Vaz, & Matos, 2006; Bhat et al., 2011). The noise levels experienced are dependent on the type of treatment being performed and the equipment used. Rather than being continuous in nature, the noise emitted during dental treatment is intermittent allowing time for the ear to rest, resulting in less damage to the cochlea hair cells (Kircher, 2003; Kircher et al., 2012).

In the 1960s there was an awareness of the noise levels produced by equipment in dental clinics and efforts have been made to produce quieter equipment (Zubick, Tolentino, & Boffa, 1980). In recent years the developments in the technology of dental equipment have produced considerable reduction in the noise emitted from equipment. The sound pressure levels generated by modern suction tubes, turbines, ultrasonic scalers and micromotor hand pieces are generally below 85 dBA (Messano &

Petti, 2012). Older worn, frequently sterilized equipment and equipment that is not regularly maintained may produce noise levels greater than 85 dBA and up to 100 dB and therefore, may potentially cause hearing damage (Fernandes et al., 2006; Mervine, 2007; Messano & Petti, 2012). This is particularly important when looking at noise levels in dental schools as the equipment in these institutions although well maintained is often old and well worn (Bhat et al., 2011; Messano & Petti, 2012).

There have been very few studies looking at the harmful effects of the ultrasonic frequency range. Studies using animals have revealed damage to the organ of corti and vestibular dysfunction after exposure to ultrasonic stimuli (Barek, Adam, & Motsch, 1999). Although the human ear does not generally perceive frequencies above 20 kHz they are still thought to damage hearing due to the production of sub-harmonics (Barek et al., 1999; Trenter & Walmsley, 2003; Bhat et al., 2011). These sub-harmonics are thought (Canadian Department of National Health and Welfare, Guidelines for the Safe Use of Ultrasound, 1991) to be generated in the ear itself or by a non-linear interaction when energy from the ultrasound is scattered at an air-water interface (Bhat et al., 2011; Canadian Department of National Health and Welfare, Guidelines for the Safe Use of Ultrasound, 1991). The sub-harmonics are perceived as high-pitched squeaky sounds. Temporary threshold shifts and some permanent threshold shift of 2-5 dB in the 13-17 kHz region have been reported after exposure to ultrasonic equipment (Barek et al., 1999).

Sorainen and Rytönen (2002) evaluated the noise spectra of air turbine and micromotor handpieces during patient treatment in 1/3-octave bands up to 80,000 Hz. The noise level of both the air turbine and the

micromotor were observed to be most powerful in the 1/3-octave band of 40,000 Hz where the levels ranged from 83-89 dB and 81-84 dB respectively. However, when these instruments were used during the treatment sound pressure level measurements revealed a L_{Aeq} of 76 dBA, which is acceptable by ISO 1999 standards, and therefore posed no risk to hearing thresholds. The authors of the study noted that although the ultrasonic levels were below the American Conference of Government Industrial Hygienists (ACGIH) limits of 105-115 dB in the 1/3-octave bands of 20,000 – 50,000 Hz, ultrasonic scalers were not used during the measurements (Sorainen & Rytönen, 2002).

The use of ultrasound scalers has been an acceptable alternative to hand scalers for the removal of dental calculus since the late 1950s. Although a valuable tool in the prevention of periodontal disease its use may potentially result in auditory damage for both the client and the clinician. The risk to the clinicians hearing as mentioned earlier is thought to be due to airborne sub-harmonics. Trenter & Walmsley (2003) reviewed the available literature and concluded with respect to the clinician, “the ultrasonic scaler has been shown to cause no permanent harm to hearing through airborne noise.”

2.1.5.2 Orthopaedics and Hearing Loss

There is concern that high environmental noise levels in the health industry may be responsible for NIHL in healthcare workers. It is reported that orthopaedic staff experience the highest prevalence of hearing-associated problems, due to the use of noisy high-powered tools during orthopaedic surgery and fracture treatment (Messano & Petti, 2012).

Many studies have looked at the noise levels in orthopaedic theatres and the risk to the orthopaedic staff. It has been reported that noise levels in

the operating room routinely exceed 100 dB and are occasionally in excess of 120 dB (Marsh, Jellicoe, Black, Monson, & Clark, 2011). A study by Kamal in 1982 showed a correlation between exposure time and “early but definite changes” in the hearing thresholds in around 50% of staff working in orthopaedic theatres. It was determined that the major source of noise in the orthopaedic theatres was the air drill and the cast saw (Kamal, 1982).

Noise levels produced by orthopaedic instruments have been measured 95 dBA to 106 dBA (Holmes, Goodman, Hang, & McCorvey, 1996). Willett, 1991, measured the noise levels at the operators’ ear produced by orthopaedic drills and saws commonly used at that time and found them to be between 90 dBA and 100 dBA. A more recent study by Siverdeen, Ali, Lakdawala and McKay, 2008, reported similar findings; the mean noise levels generated by the saws, drills, K-wire drills and hammers were 95 dBA, 90 dBA, 85 dBA 65 dBA respectively, however these levels were measured at the patients ear not the operators’ ear (Siverdeen, Ali, Lakdawala, & McKay, 2008).

Although these levels are potentially hazardous most of this equipment is only in use for brief periods during orthopaedic surgery. For example, the mean duration of use of powered orthopaedic equipment during a total hip replacement is about 190 seconds and 375 seconds for a total knee replacement (Willett, 1991) and the LAeq8h for one total knee replacement has been measured at 59.6 to 66.9 dBA (Sydney, Lepp, Whitehouse, & Crawford, 2007).

Few studies have looked at the noise levels present in cast clinics. Marsh, Jellicoe, Black, Monson & Clark, 2011, measured the noise levels in seven adult “cast clinics” and seven paediatric “cast clinics” and found LAeq8h levels of 76.6 dBA and 75.9 dBA and mean peak noise levels of 140.0 and

140.7, respectively. Marsh et al, 2011, concluded that although mean noise levels were within recommended safety limits, peak noise levels in all clinics, which exceeded recommended safety limits, were potentially hazardous (Marsh et al., 2011).

2.2 Summary

Based on the literature, there was strong evidence to support the cause effect relationship between NIHL and noise exposure, either occupational or recreational. Those working in the health industry, especially in dentistry and orthopaedics have been identified as individuals at risk of NIHL because of the use of drilling and sawing equipment. As well as the noise-related auditory effects many non-auditory noise-related health effects have also been identified. Noise-related health affects impact on the social and economic status of the individual worker and the wider community. Individual worker safety may also be compromised. Although many studies have been performed internationally there is little information available on the noise levels in New Zealand dental and orthopaedic clinics.

2.3 References

- Albera, R., Lacilla, M., Piumetto, E., & Canale, A. (2009). Noise-induced hearing loss evolution: influence of age and exposure to noise. *European Archives of Oto-rhino-laryngology*, 267, 665-671.
- Attias, J., Bresloff, I., Joachims, Z., & Ising, H. (1998). Prophylactic effect of magesium in noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 271-279). London: Whurr Publishers Ltd.
- Australia Standards/New Zealand Standards. (2000). *Acoustics—Recommended design sound levels and reverberation times for building interiors*. (No. AS/NZS 2107:2000).
- Babisch, W. (2003). Stress hormones in the research on cardiovascular effects of noise. *Noise & Health*, 5(18), 1-11.
- Babisch, W. (2011). Cardiovascular effects of noise. *Noise & Health* (Vol. 13, pp. 201).
- Barek, S., Adam, O., & Motsch, J. F. (1999). Large band spectral analysis and harmful risks of dental turbines. *Clinical Oral Investigation*, 3, 59-54.
- Bhat, P., Jyothi, C., Kadanakuppe, S., & Ramegowda, C. (2011). Assessment of noise levels of the equipments used in the dental teaching institution, Bangalore. *Indian Journal of Dental Research*, 223, 424-431.
- Bies, D. A., & Hansen, C. H. (1988). *Engineering Noise Control*. London: Unwin Hyman Ltd.
- Broadbent, D. E. (1979). Human performance and noise. In C. M. Harris (Ed.), *Handbook of noise control*. New York: McGraw-Hill, Inc.
- Canadian Department of National Health and Welfare. *Guidelines for the Safe Use of Ultrasound*. 1991. Obtained from http://www.hc-sc.gc.ca/ewh-semt/pubs/radiation/safety-code_24-securite/index-eng.php
- Carmen, R. (1999). Tinnitus and hearing loss. *Occupational Health and Safety*, 68(10), 154-158.
- Carter, L., Gilliver, M., Macoun, D., Rosen, J., & Williams, W. (2012). Music to whose ears? The effect of social norms on young people's risk perceptions of hearing damage resulting from their music listening behaviour. *Noise and Health* (Vol. 14, pp. 47).

- Fernandes, C. S., Carvalho, A. P. O., Gallas, M., Vaz, P., & Matos, P. A. (2006). Noise levels in dental schools. *European Journal of Dental Education*, 28, 32-37.
- Feston, J. M., George, E. L. J., Goverts, S. T., & Hougast, T. (2010). Measuring the effects of reverberation and noise on sentence intelligibility for hearing impaired listeners *Journal of Speech, Language, and Hearing Research* (Vol. 53, pp. 1429).
- Feuerstein, J., & Marshall, C. (2009). Noise exposure and issues in hearing conservation. In J. Katz, L. Medwetsky, R. Burkard & L. Hood (Eds.), *Handbook of Clinical Audiology*. Baltimore: Lippincott Williams & Wilkins.
- Fidell, S. (1979). Community Response to Noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.
- Gastmeier, W., & Aitken, D. R. (1999). Reverberation in Gymnasias. *Canadian Acoustics*, 27(4), 3-7.
- Gijbels, F., Jacobs, R., Princen, K., Nackaerts, O., & Debruyne, F. (2006). Potential occupational health problems for dentists in Flanders, Belgium. *Clinical Oral Investigation*, 10, 8-16.
- Glorig, A., & Nixon, J. (1962). Hearing loss as a function of age. *Laryngoscope*, 72, 1590-1610.
- Guidelines for the safe use of ultrasound*. (1991). Ottawa, Canada: Canadian Communication Group - Publishing.
- Haller, A. K., & Monygomery, J. K. (2004). Noise-induced hearing loss in children. *Teaching Exceptional Children*, 36(4), 22-27.
- Hansen, C. (2005). *Noise Control from Concept to Application*. London: Taylor & Francis.
- Harris, C. M. (1979). Sound and sound levels. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill Inc.
- Henderson, D., Subramanian, M., & Boettcher, F. A. (1993). Individual susceptibility to noise-induced hearing loss: an old topic revisited. *Ear & Health*, 14(3), 152-168.
- Holmes, G. B., Goodman, K. L., Hang, D. W., & McCorvey, V. M. (1996). Noise levels in orthopedic instruments and their potential health risks. *Orthopedics* 19(1), 35-37.

- Hood, L. J. (1998). *Clinical Applications of the Auditory Brainstem Response*. New York: Delmar Cengage Learning.
- Iannace, G., Lembo, P., Maffei, L., & Nataletti, P. (2006). *Acoustical conditions and noise exposure inside school gymnasia and swimming pools*. Paper presented at the Euronoise, Tempra: Finland.
- Ising, H., Babisch, W., & Kruppa, B. (1999). Noise-induced endocrine effects and cardiovascular risk. *Noise & Health*, 1(4), 37-48.
- ISO 1999-1990 Acoustics - Determination of Occupational Noise Exposure Estimation of Noise-induced Hearing Impairment. Geneva: International Organization for Standardization.
- Job, R. F. S. (1996). The influence of subjective reactions to noise on the health effects of the noise. *Environmental International*, 22(1), 93-104.
- Kamal, S. A. (1982). Orthopaedic theatres: a possible noise hazard? *The Journal of Laryngology and Otology*, 96, 985-990.
- Kircher, D. B. (2003). Noise-induced hearing loss. *Journal of Occupational & Environmental Medicine*, 45(6), 579-581.
- Kircher, D. B., Evenson, E., Dobie, R. A., Rabinowitz, P. M., Crawford, J., Kopke, R., & Hudson, T. W. (2012). Occupational noise-induced hearing loss: ACOEM task force on occupational hearing loss. *Journal of Occupational & Environmental Medicine*, 54(1), 106-108.
- Kryter, K. D. (1972). Non-auditory effects of environmental noise. *American Journal of Public Health* 62(3), 389-398.
- Kua, H. W., Lee, S. E., & Mahbub, A. S. (2010). A total building performance approach to evaluating building acoustics performance. *Architectural Science Review*, 53(2), 213.
- Landstrom, U., & Akerlund, E. (1995). Exposure levels, tonal components, and noise annoyance in working environments. *Environmental International*, 21(3), 265-275.
- Lipscomb, D. M. (1994). *Hearing Conservation in industry, schools, and the Military*. San Diego, CA: Singular Publishing Group.
- Lookwood, A. H., Salvi, R. J., & Burkard, R. F. (2002). Tinnitus. *The New England Journal of Medicine*, 347(12), 904-910.

- Marsh, J. P., Jellicoe, P., Black, B., Monson, R. C., & Clark, T. A. (2011). Noise levels in adult and pediatric cast clinics. *The American Journal of Orthopedics*, 40(7), E122-E124.
- Melnick, W. (1991). Human temporary threshold shift and damage risk. *The Journal of the Acoustical Society of America*, 90(1), 147-153.
- Mervine, R. (2007). Noise-induced hearingloss in dental offices. *Dental Economics*, 97(1), 1-7.
- Messano, G. A., & Petti, S. (2012). General dental practitioners and hearing impairment. *Journal of Dentistry*, 40, 821-828.
- Miller, J. D. (1974). Effects of noise on people. *The Journal of the Acoustical Society of America*, 56, 729-764.
- Molino, J. A. (1979). Annoyance and Noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.
- OSH. (2002). *Approved code of practice for the management of noise in the workplace*. Wellington: Occupational Safety and Health Service.
- Parham, K., Gates, G., Dobie, R. A., McKinnon, R., & Backous, D. (2010). Challenges and opportunities in presbycusis. *Otolaryngology-Head and Neck Surgery*, 143, 31.
- Patel, R., & Schell, K. W. (2008). The influence of linguistic content on the Lombard effect. *Journal of Speech, Language, and Hearing Research*, 51(1), 209-220.
- Peterson, A. P. G. (1979). Noise measurements: instruments. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill, Inc.
- Prasher, D. (1998). Factors influencing susceptibility to noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 125-131). London: Whurr Publishers Ltd.
- Pyykko, I., Starck, J., Toppila, E., & Kaksonen, R. (1998). Ageing as a major confounding factor in noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 157-163). London: Whurr Publishers Ltd.
- Raney, J. P., & Cawthorn, J. M. (1979). Aircraft noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.

- Royster, J. D., Royster, L. H., & Killion, M. C. (1991). Sound exposures and hearing thresholds of symphony orchestra musicians. *The Journal of the Acoustical Society of America*, 89(6), 2793-2802.
- Schroeder, M. R. (1980). Acoustics in human communications: Room acoustics, music, and speech. *Journal of the Acoustical Society of America*, 68(1), 22-29.
- Setcos, J. C., & Mahayuddin, A. (1998). Noise levels encountered in dental clinical and laboratory practice. *International Journal of Prosthodontics*, 11, 150-157.
- Sharland, I. (1972). *Woods Practical Guide to Noise Control*. London: Woods of Cholchester Limited.
- Shepard, D., Welch, D., Dirks, K. N., & Mathews, R. (2010). Exploring relationships between noise sensitivity, annoyance and health-related quality of life in a sample of adults exposed to environmental noise. *International Journal of Environmental Research and Public Health*, 7, 3579-3594.
- Siverdeen, Z., Ali, A., Lakdawala, A. S., & McKay, C. (2008). Exposure to noise in orthopaedic theatres – do we need protection? *International Journal of Clinical Practice*, 62(11), 1720-1722.
- Smith, A. (1991). A review of the non-auditory health effects of noise on health. *Work & Noise*, 5(1), 49-62.
- Sorainen, E., & Rytönen, E. (2002). Noise level and ultrasound spectra during burring. *Clinical Oral Investigation*, 6, 133-136.
- Speaks, C. E. (2005). *Introduction to Sound: Acoustics for Hearing and Speech Sciences*. USA: Thomson Delmar Learning.
- Spreng, M. (2000). Possible health effects of noise induced cortisol increase. *Noise & Health*, 2(7), 59-63.
- Stach, B. A. (1998). *Clinical Audiology: an Introduction*. San Diego: Singular Publishing Group, Inc.
- Stach, B. A. (2003). *Comprehensive Dictionary of Audiology Illustrated* (2nd ed.). New York: Delmar Cengage Learning.
- Stansfeld, S. A., & Matheson, M. P. (2003). Noise pollution: non-auditory effects on health. *British Medical Bulletin*, 68, 243-257.

- Starck, J. (1998). How should different susceptibility factors be evaluated. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 121-123). London: Whurr Publishers Ltd.
- Suter, A. H., & Berger, E. H. (2002). *Hearing conservation manual* (4 ed.). Milwaukee.
- Sydney, S. E., Lepp, A. J., Whitehouse, S. L., & Crawford, R. W. (2007). Noise exposure due to orthopedic saws in simulated total knee arthroplasty surgery. *The Journal of Arthroplasty*, 22(8), 1193-1197.
- Trenter, S. C., & Walmsley, A. D. (2003). Ultrasonic dental scaler: associated hazards. *Journal of Clinical Periodontology*, 30, 95-101.
- van Dijk, F. J. H. (1990). Epidemiological research on non-auditory effects of occupational noise exposure. *Environmental International*, 16, 405-409.
- Venema, T. H. (2006). *Compression for Clinicians* (2nd ed.). Canada: Thompson Delmar Learning.
- Ward, W. D. (1971). Presbycusis, sociocusis and occupational noise-induced hearing loss. *Proceedings of the Royal College of Medicine*, 64, 200-203.
- Webster, J. C. (1979). Effects of Noise on Speech. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill, Inc.
- WHO. (1997). Prevention of noise-induced hearing loss. Geneva: World Health Organisation.
- WHO. (1999). Constitution of the World Health Organization
- WHO. (2011). Burden of disease from environmental noise. Obtained from http://www.euro.who.int/_data/assets/pdf_file/0008/.../e94888.pdf
- WHO. Night Noise Guidelines for Europe. Obtained from <http://www.euro.who.int/document/e92845.pdf>
- Willett, K. M. (1991). Noise-induced hearing loss in orthopaedic staff. *The Journal of Bone and Joint Surgery*, 73 B(1), 113-115.
- Williams, W., & Purnell, J. (2010). The statistical distribution of expected noise level output from commonly available personal stereo players. *Acoustics Australia*, 38, 119-122.

- Willich, S. N., Wegscheider, K., Stallmann, M., & Keil, T. (2006). Noise burden and the risk of myocardial infarction *European Heart Journal*, 27, 276-282.
- Yost, W. A. (2000). *Fundamentals of Hearing: An Introduction* (4th ed.). San Diego: Academic Press, Inc.
- Yost, W. A., & Neilson, D. W. (1997). *Fundamentals of Hearing: An Introduction*. USA: Holt, Rinehart and Winston.
- Zollinger, S. A., & Brumm, H. (2011). The Lombard effect. *Current Biology*, 21(16), 614-615.
- Zubick, H. H., Tolentino, A. T., & Boffa, J. (1980). Hearing loss and the highspeed dental drill. *American Journal of Public Health*, 70(6), 633-635.

3

Methodology

3.1 Acoustic Assessment of the Healthcare Clinics

This project included two stages of data collection and analysis. The first stage of the study identified the acoustic characteristics of rooms used for clinical procedures at three dental clinics while the second stage identified the acoustic characteristics of an orthopaedic clinic.

The acoustic measurement in stage one were:

- I. The ambient noise level and reverberation time of each dental clinic while not in use;
- II. Spectral analysis of the noise emissions from the dental equipment;
- III. The noise level during a typical working day measured at the clinician's ear.

The acoustic measurements in stage two were:

- I. Spectral analysis of the noise emissions from the cast saw
- II. Background noise measurements in the orthopaedic cast clinic; and

- III. The noise level during a typical working day measured at the ear of an orthopaedic nurse.

3.2 Methodology

This section records the instrumentation and procedure in the study and describes the measurements made.

3.2.1 Instrumentation

The follow equipment was used to measure the ambient noise levels, spectral analysis and reverberation times.

3.2.1.1 Hand-held Analyzer Bruel &Kjaer – 2250 (B&K 2250)

The B&K 2250 is a hand-held sound level meter used in conjunction with Frequency Analysis Software – BZ7223 used for measuring and analyzing sound. The software enables the B&K 2250 to make real time measurement in octave bands centred at 8 Hz to 16 kHz and 1/3 octave bands centred at 6.3 Hz to 20 kHz. The B&K 2250 is capable of recording a comprehensive range of time measured parameters including Equivalent Continuous Sound Levels (L_{eq}), Peak Sound Levels (L_{peak}), Maximum Time weighted Sound Levels (L_{max}) and Minimum Time-weighted Sound Levels (L_{min}).

3.2.1.2 Modular Precision Sound Analyzer Bruel & Kjaer – 2260 (B&K 2260)

The B&K 2260 is a hand-held sound level meter used in conjunction with Sound Analysis Software – BZ7201 used for measuring and analyzing sound noise and vibration. The software enables the B&K 2260 to make real time measurement in octave bands centred at 8 Hz to 16 kHz and 1/3 octave bands centred at 6.3 Hz to 20 kHz. The B&K 2260 is capable of measuring and analyzing numerous discrete noise parameters and providing statistical and frequency data.

The B&K 2260 has a built-in noise generator, which was used to generate pink noise during the measurement of reverberation times.

3.2.1.3 Sound Level Calibrator Bruel & Kjaer – 4231 (B&K 4231)

A hand-held sound source for calibration of sound meters and other sound measurement equipment used for calibrating Bruel & Kjaer sound measuring equipment with 1 inch and 1/2 inch microphones. The B&K 4231 uses a calibration frequency of 1000 Hz and a calibration pressure of 94 ± 0.2 dB re 20Pa.

3.2.1.4 JBL Powered Speaker – EON Power 10

The EON Power 10 is a lightweight speaker system that uses a 60-watt power amplifier for low frequencies and a 25-watt power amplifier for high frequencies both with 0.1 % total harmonic distortion. The EON Power 10 has

a frequency range (-10 dB) from 60 Hz to 18 kHz and a frequency response (-3 dB) from 80 Hz to 16 Hz.

The following piece of equipment was used to record daily noise dose measurements at the participants' ear.

3.2.1.5 Noise Dose Meter Bruel & Kjaer – 4436 (B&K 4436)

The B&K 4436 is a noise dose meter used for measuring Sound Exposure (Pa^2h) and Daily Personal Dose Exposure Level ($L_{EP,d}$). The B&K 4436 satisfies a wide range of International Noise Exposure Standards. The B&K 4436 has a sampling rate of 16 times per second (16 Hz) and the distribution and cumulative distribution are measured in 1 dB intervals.

3.2.2 Instrumentation Setup and Procedure

The instrumentation setup and procedure used to obtain ambient noise levels, reverberation times and daily noise dose measurements were as follows:

3.2.2.1 Ambient Noise Levels and Reverberation Time

Each dental clinic was measured, using a standard builders' tape measure, and the dimensions of the clinic along with measurements of the main fitting and fixtures recorded. A record was taken of the surface materials used in the clinic for use later in Sabine calculations.

Each clinic was then assessed to determine the possible sites for the noise source and measurement microphone. Because of the limited space in

the dental clinic a limited number of observation positions were used. Two speaker positions and two microphone positions were used giving a total of four recordings for each dental clinic.

The ambient noise level of the clinic was measured while the clinic was not in use. The B&K 2250 sound analyzer was calibrated using the B&K 4231 calibrator before each day's measurements. The background noise level was sampled for 10 seconds, 3 measurements were taken at each clinic to obtain an average noise level. In the orthopaedic cast clinic, background noise measurements were obtained using the B&K 2260 sound analyzer in a central location within the cast clinic.

The B&K 2250 sound analyzer was positioned at least 1 metre from major reflecting surfaces, such as, walls or windows. The output of the B&K 2250 noise generator was connected to the amplifier, which was coupled to the JBL speaker. The B&K 2250 was set as follows: "escape time" of 10 seconds to allow the testers to vacate the dental clinic before the testing began; "build-up time" of 5 seconds to allow a steady sound pressure level to build up before decay measurements began being recorded; and "decay time" of 5 second to allow for the sound pressure level in the clinic to fall by at least 20/30 dB to obtain the RT20 and RT30 measurements before the B&K 2250 completed the calculation of the reverberation time, the RT60.

The JBL speaker was mounted at a level 1.2 metres above the floor at approximately ear level of the clinician. Using the JBL speaker the pink noise was generated in the clinical environment. This was then analyzed with the aid of the B&K 2250 to determine the decay time, that is, for the level of the noise to drop by 20/30 dB. The B&K 2250 extrapolates from this data the time required for the noise to drop by 60 dB, that is, the RT60. The RT60 was

measured over a frequency range of 0.08 kHz to 10 kHz. A diagram of the room layout of each clinic is shown in Appendix 5.

3.2.2.2 Spectral Analysis of Dental Equipment Noise

Measurements for spectral analysis were taken at each dental clinic and in the orthopaedic cast clinic. The B&K 2250 spectral analyzer was calibrated, according to the manufacturer's instructions, using a B&K 4231 calibrator prior to taking measurements at each clinic. Measurements using the B&K 2250 spectral analyzer were made by placing the microphone of the analyzer within 2 cm of the equipment being investigated during operation and at the ear of the clinician. As the clinician was working on a patient during recording, care was required so as to not cause any interference. A 10 second noise sample was recorded for the equipment during clinical use. The noise sample was analyzed using the proprietary software.

3.2.2.3 Noise Dose

The personal noise dosimeter (B&K 4436) was used to assess the noise level during the working day on three separate occasions at each dental clinic and on six occasions in the orthopaedic cast clinic. Daily calibration was performed prior to commencement of recordings and after completion of recordings according to the manufacturer's instructions.

At the beginning of the working day the body of the dosimeter was clipped to the belt or placed in a pocket at the waist of the participant. The microphone tubing was attached to the shoulder of the participant's tunic so that the microphone was positioned within 10 cm of the clinician's ear. The

dosemeter was set to record and locked so that the settings would not be altered if accidentally bumped.

The dosimeter was removed, unlocked and turned off at the end of the day. The B&K 4436 stores only the information from the current measurements therefore it was necessary to make a record of each day's measurements. Three dosimeter measurements were made at each dental clinic and six measurements were made in the orthopaedic cast clinic.

4

Results

4.1 Stage One: Dental Clinics

The experimental measures obtained in the first stage of the study were the reverberation time (in seconds), ambient sound pressure level (in dBA), the type and area of the surface materials, frequency analysis of noise sources, and the daily noise dose (in L_{eq8h}). The reverberation time was measured from 80 Hz to 10 kHz, however, the frequency range of 500 Hz to 5 kHz is of particular note as this encompasses the speech frequency range. The average reverberation time was calculated from measurements made at two microphone positions. The reverberation time, as previously described, was the time taken for the sound to drop 60 dB below its original level. Long reverberation time causes speech to become less intelligible and higher background noise levels are present (Sharland, 1972).

4.1.1 Room Measurements

Measurements were made of each dental surgery. Room dimensions were noted along with the main fittings and fixtures for use in calculating the reverberation time using the Sabine equation, see Appendix 5.

4.1.1.1 Room Measurements: Clinic 1

Clinic 1 was a small room situated on the ground floor at the rear of the building so was therefore away from any traffic noise, which in any case was minimal as the building was located in a quiet street. There was an air conditioning unit within the room and the room was adjacent to the preparation/sterilizing room. At the time of measurement there was a lot of activity in the preparation room and the air conditioning unit was operating.

The average ambient noise level in clinic 1 was 40 dBA, which falls within the recommended ambient noise levels in Australian Standard/New Zealand Standard AS/NZS 2107:2000 of 40-45 dBA.

The reverberation time measurements for clinic 1 can be seen below in Figure 4.1 along with the calculated reverberation times from the Sabine calculations in Appendix 5. The measured reverberation times ranged from 0.27s to 0.47s, which fall below the maximum recommended reverberation time as set out in the Australian Standard/New Zealand Standard AS/NZS 2107:2000 of 0.60s. This would suggest there would be little or no effect on speech intelligibility from the reverberant noise.

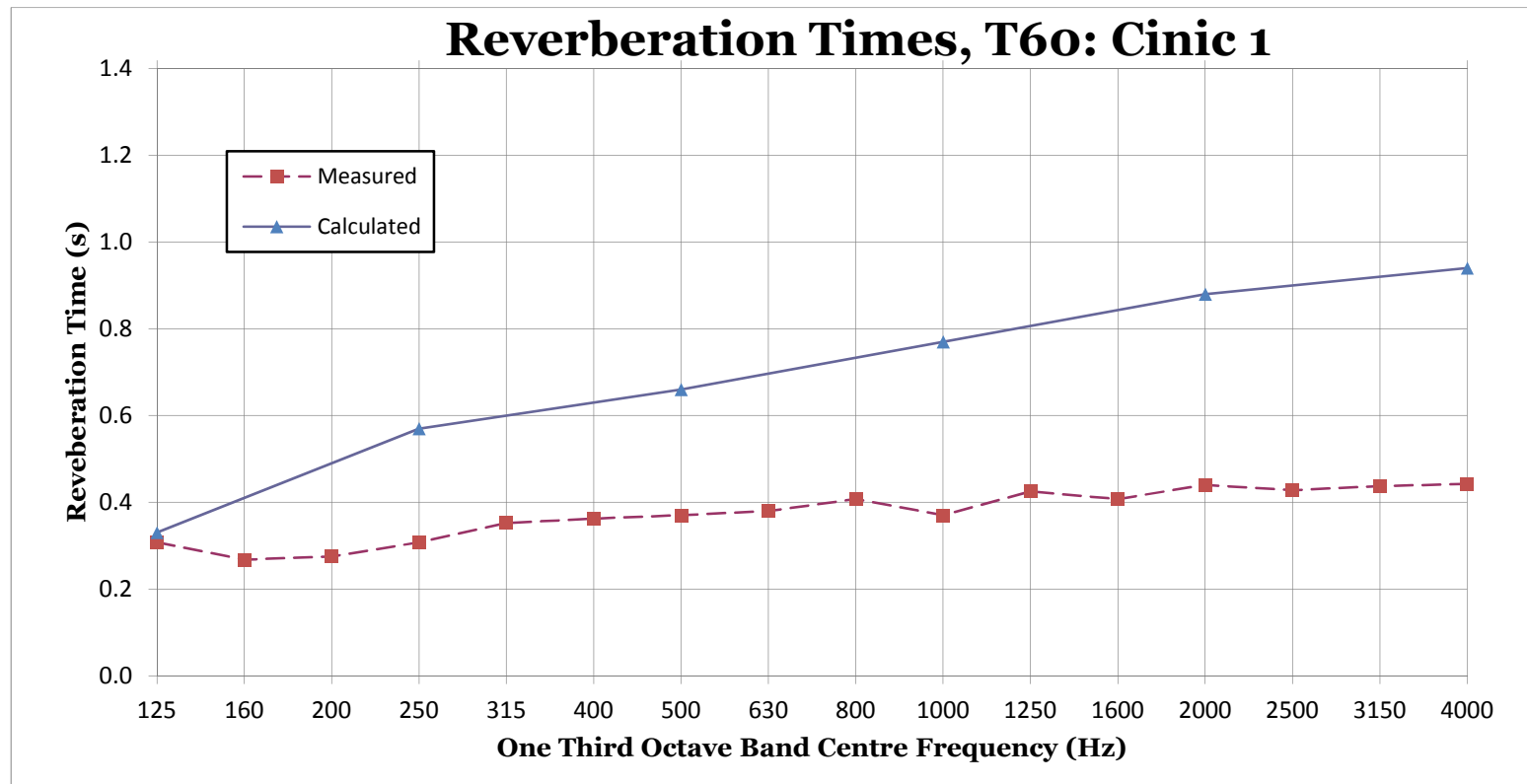


Figure 4.1: Reverberation Time: Clinic 1. The measured reverberation time (RT60) extrapolated from RT20 times and the and calculated reverberation time for Clinic 1 at the centre frequency of 1/3-octave bands.

4.1.1.2 Room Measurements: Clinic 2

Clinic 2 was a large clinic situated on the first floor at the front of the building and was exposed to traffic noise from a busy main road. The only window in the room faced the road. The window was double-glazed. There was an air conditioning unit within the room, which at the time of measurement was operating.

The average ambient noise level in clinic 1 was 38 dBA, which falls below the recommended ambient noise levels in Australian Standard/New Zealand Standard AS/NZS 2107:2000 of 40-45 dBA.

The reverberation time measurements for clinic 2 can be seen below in Figure 4.2 along with the calculated reverberation times from the Sabine calculations in Appendix 5. The reverberation times ranged from 0.52s to 1.36s in the low frequencies below 250 Hz, with a range from 0.60s to 0.98s at frequencies between 250 Hz and 5000 Hz. The Australian Standard/New Zealand Standard AS/NZS 2107:2000 sets a maximum recommended reverberation time in medical rooms of 0.60s. The reverberation times in Clinic 2 do not meet this recommendation; and would likely result in a reduction of speech intelligibility due to reverberant noise.

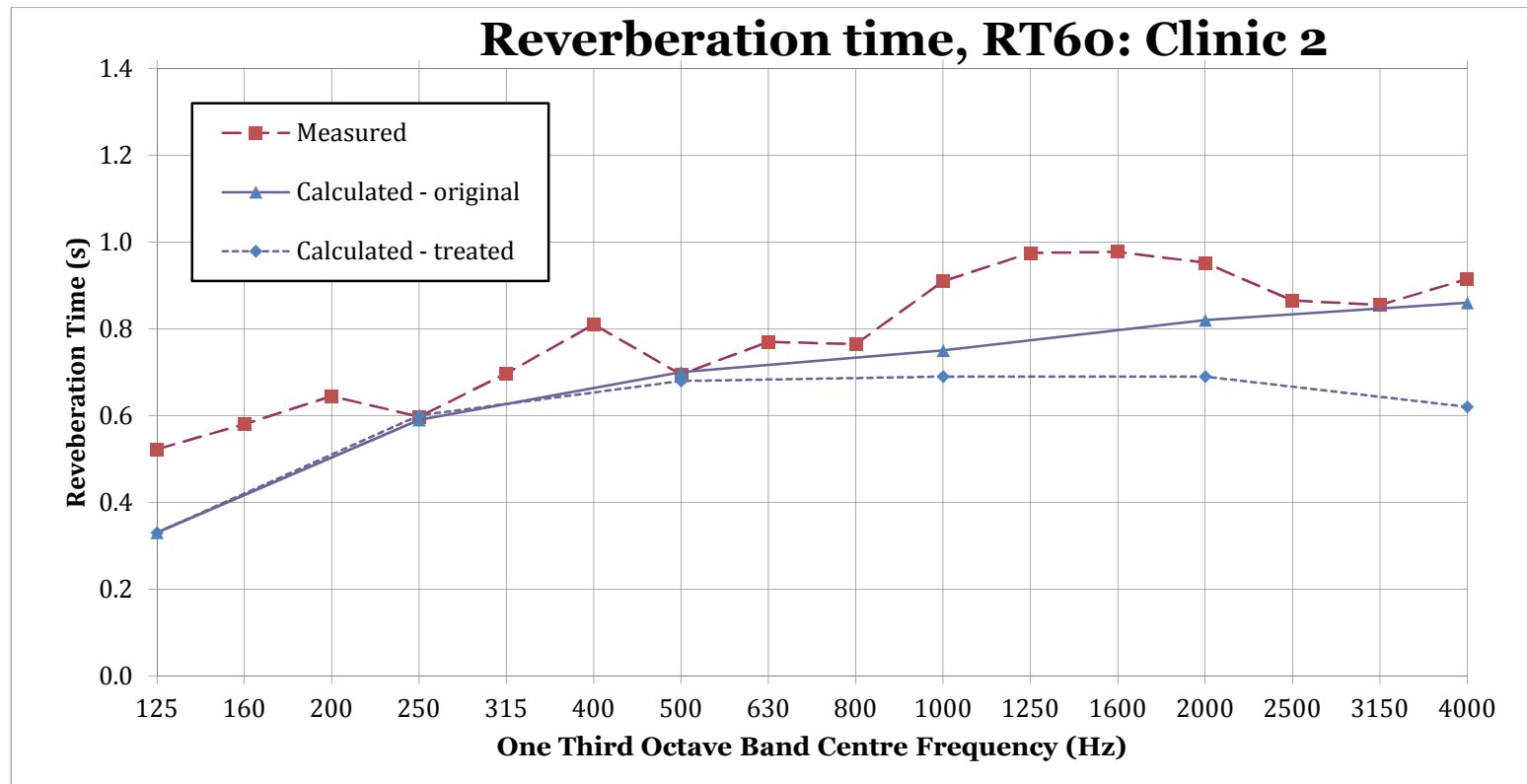


Figure 4.2: Reverberation Time: Clinic 2. The measured reverberation time (RT60) extrapolated from RT20 times and the calculated reverberation time for Clinic 2 at the centre frequency of 1/3-octave bands.

4.1.1.3 Room Measurements: Clinic 3

Clinic 3 was a large clinic situated on the ground floor at the rear of the building so therefore was away from traffic noise. The building was located on a busy street. There was carpet on the floor in the clinic that covered about half of the floor space. There was an air conditioning unit within the room, which at the time of measurement was operating. Only two samples were taken at this clinic, as the room was required for the treatment of patients.

The average ambient noise level in clinic 3 was 36 dBA, which falls within the recommended ambient noise levels in Australian Standard/New Zealand Standard AS/NZS 2107:2000 of 40-45 dBA.

The reverberation time measurements for clinic 3 can be seen below in Figure 4.3. The reverberation times ranged from 0.27s to 0.51s, which fall below the maximum recommended reverberation time in Australian Standard/New Zealand Standard AS/NZS 2107:2000 of 0.60s. This would suggest there would be little or no effect on speech intelligibility from reverberant noise.

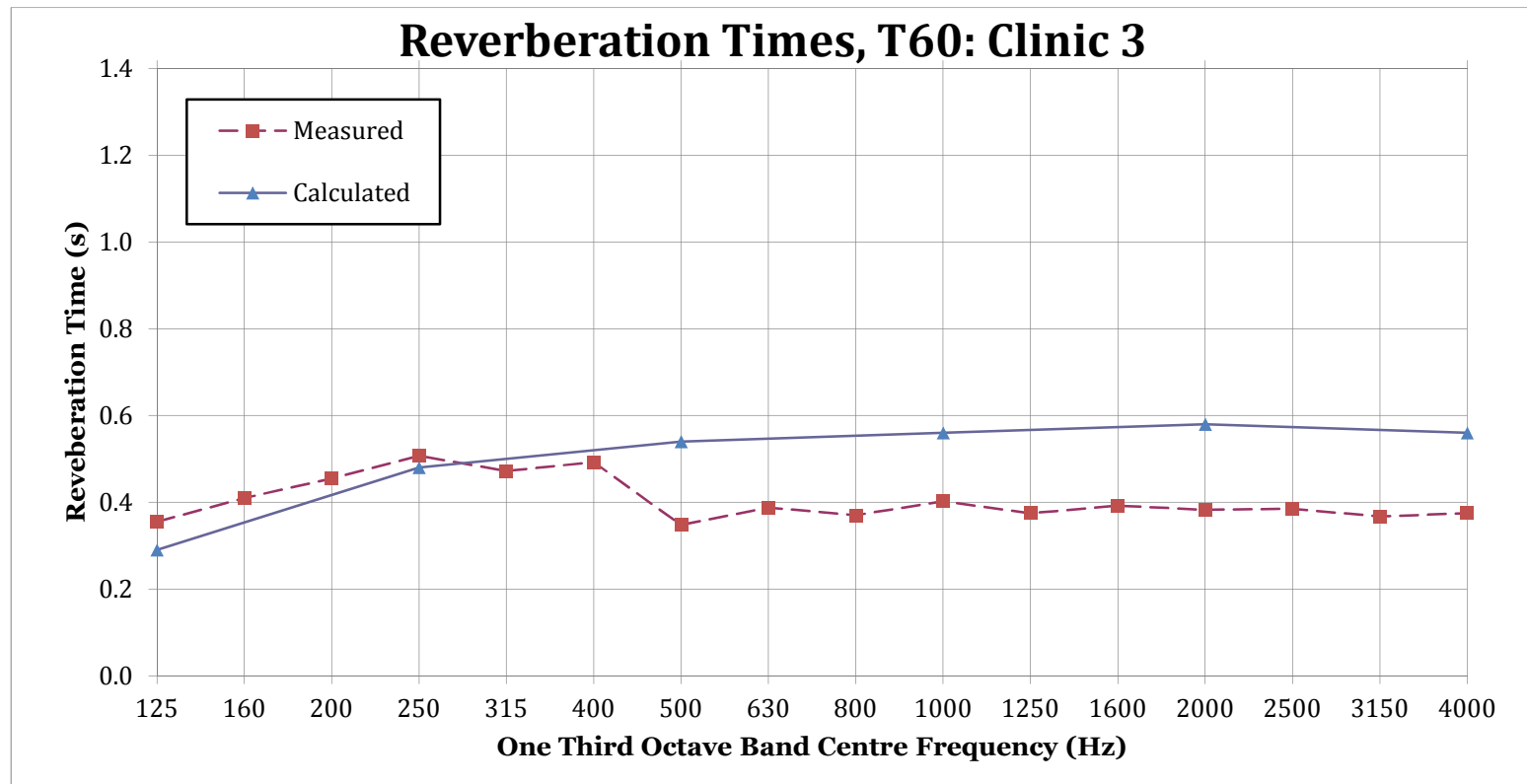


Figure 4.3: Reverberation Time: Clinic 3. The measured reverberation time (RT60) extrapolated from RT20 times and the calculated Reverberation times for Clinic 3 at centre frequencies of 1/3-octave bands.

4.1.1.4 Discussion: Ambient Noise Level and Reverberation Time

Clinic 1 had the highest ambient noise level due in part to its proximity to the preparation room but also to its smaller size. A smaller room size results in an increased number of sound reflections therefore increasing the reverberation time resulting in raised sound pressure levels. This can be seen in the Sabine calculations in Appendix 5.

Although Clinic 2 and Clinic 3 are similar in size they show a marked difference in ambient noise level and reverberation time. These differences probably at least in part are due to difference in floor coverings. The entire floor surface in Clinic 2 was covered with vinyl while approximately half the floor surface in Clinic 3 was covered with acoustically more absorbent carpet.

4.1.2 Spectral Analysis of Dental Equipment Noise

From Figures A.4.1, A.4.2 and A.4.3, in Appendix 4, similar trends were seen in all three clinics, in that, the noise from the suction equipment raised the sound pressure level across the frequency range from 100 Hz to 12,500 Hz, with a notable peak in the 1250 Hz to 2000 Hz one-third octave bands. A similar peak was seen in the high-speed drill measurement, which can be attributed to the suction equipment operating in the background. The differences between the suction and the high-speed drill measurements at the high frequencies was likely due to the tonal characteristics of the high-speed drill, which operate at speeds of over 200,000 rpm (3333 Hz). Peaks were seen in the noise from the high-speed drills at 5000 Hz and 10,000 Hz in Clinic 1 and 4000 Hz and 8000 Hz in Clinic 2. The increase in sound pressure level in the 250 Hz to 1000 Hz one-third octave bands may be the result of

vibrations from the high-speed drill. The peaks in the ear-level measurements corresponded to peaks in the suction and high-speed drill measurements. The scaler used in Clinic 3 appeared to have similar characteristics to that of the high-speed drill up to 12,500 Hz.

Spectral analysis of the noise was performed on one-third octave bands and therefore, although peaks are apparent in the spectrum of the high-speed drill, no definite conclusions could be made. It is clear that the high-speed drill produces a significant amount of energy at the high frequencies, however, a narrow band analysis and knowledge of the high-speed drill rotational speed would be required before confirming any tonal contributions.

4.1.3 Dosimeter Noise Exposure levels in Dental Clinics

The specific daily dose measurements obtained at each clinic can be found in Appendix A.2. Table 4.1 the results obtained from measurement of the daily noise exposure at the clinicians ear in each dental surgery.

Clinic	Range L_{eq8h} (%)	Average L_{eq8h} (%)
1	3 – 12	7.0
2	4 – 5	4.7
3	3 – 4	3.7

Table 4.1: Daily Noise Dose Measurements. The table shows the daily noise dose measurements from dental surgeries 1, 2, and 3, and from the orthopaedics cast clinic as a percentage of L_{eq8h} 85 dBA (%).

L_{eq8h} (dBA)	% daily dose (%)	Acceptable exposure time (hrs/day)
70	3.13	-
73	6.25	-
76	12.5	-
79	25	-
82	50	16
85	100	8
88	200	4
91	400	2
94	800	1

Table 4.2: Noise Exposure and Maximum Permissible Exposure Time. The eight-hour equivalent continuous noise level (L_{eq8h}), in dBA, represented as a percentage of a daily dose and maximum exposure time in hours per day.

The results shown in Table 4.1 show that dental clinicians who participated in this study were exposed to on average 3.7 to 7.0% of a daily dose of noise using an L_{eq8h} of 85 dBA. From Table 4.2 below it can be seen that this is the equivalent to an L_{eq8h} of 70 – 73 dBA. This would suggest that those working in the dental clinics are not at risk of NIHL but may still be at risk of non-auditory health effects of noise exposure.

It is important to note that the daily noise dose measure at clinic 1 on day three may be a bit higher than would normally be expected. The researcher had commented to the clinician that the previous two noise dose recordings were quite low, to which the clinician indicated that he would endeavor to have a noisier day. If the result for day 3 is remove the average for clinic 1 range of L_{eq8h} would be from 3 to 6 % and the average would then be 4.5 %.

4.2 Stage Two: Orthopaedic Cast Clinic

The experimental measures obtained in the second stage of the study were the background sound pressure levels (in dBA), noise levels with cast cutting saws in operation, and the daily noise dose (in L_{eq8h}).

4.2.1 Background Noise Measurements

The orthopaedic cast clinic was a large 8-bedded room situated on the first floor in the middle of a 6-storied building. There were no windows in the room. There were doors at the north and south end of the room that remained open. There was an air conditioning unit within the room and the room was adjacent to the preparation and storage rooms. The nurses' station was situated in an alcove at the south end of the room. Each bed was separated from the neighbouring bed by a fabric curtain.

The cast clinic has a maximum occupancy of eight patients who are usually accompanied by one or more support person. Seven or eight members of the nursing staff are present for a daytime shift during which time, up to three medical teams, each consisting of two or three members, may also be present. Up to three cast-cutting saws may be in operation at any time during the working day.

Background noise measurements were taken during a period of 30 minutes on a daytime shift from a position in the centre of the room. At the time patients occupied six of the eight beds and two medical teams were in attendance. The background noise measurements as shown in Table 4.3, range from 57 dBA to 76 dBA, well above the recommended ambient noise levels in Australian/New Zealand Standard AS/NZS 2107:2000 of 40-45 dBA.

Background Noise Levels	
Number of saws in operation	Sound pressure level (dBA)
0	57
0	61
0	61
0	65
1	70
1	70
1	72
2	76
2	76

Table 4.3: Background Noise Levels in the Orthopaedic Cast Clinic. Sound pressure levels in dBA were measured in the centre of the room during normal working activity.

Measurements were taken of the sound pressure level produced by a handheld electric cast-cutting saw typically used in the cast clinic for the cutting and removal of cast and fibreglass casts. Measurements were taken at the ear of the nurse operating the saw and at a distance approximately 2 cm for the cutting blade of the saw; these measurements can be seen in Table 4.4. The sound pressure levels produced by the cast cutting saw was 91 dBA, which has the potential to cause noise damage. However when measured at the nurse's ear the total noise level was 83 dBA, which, even if the saw were to be operated continuously over an eight-hour shift, would still fall below the allowable L_{eq8h} of 85 dBA.

Operating Noise: 1 Saw		
Position	Material being cut	Sound pressure level (dBA)
Within 2 cm of blade	Fibreglass	91
Within 2 cm of blade	Cast clinic	91
Nurse's ear	Fibreglass	83
Nurse's ear	Fibreglass	83

Table 4.4: Sound Pressure Levels of a Cast-cutting Saw. Sound pressure level measurements, in dBA, of the cast-cutting saw at a distance of about 2 cm from the cutting blade and at the ear of the nurse.

4.2.2 Daily Noise Exposure Levels in the Orthopaedic Cast Clinic

The specific daily dose measurements obtained in the orthopaedic cast clinic can be found in Appendix A.2. These results showed that the orthopaedic nurses, who participated in this study, were exposed to between 6% to 27% of a daily dose of noise using an L_{eq8h} of 85 dBA with an average of 13%. From Table 4.2 above it can be seen that this is equivalent to an L_{eq8h} of about 73 – 80 dBA. This would suggest that those working in the orthopaedic cast clinic are not at risk of NHIL but may still be at risk of non-auditory health effects of noise exposure. Nursing staff working in the cast clinic described the five days on which the measurements were taken as “quiet days” and that on noisier days they would often need to leave the room because of the noise levels and associated stress. Nursing staff also reported that on noisier days they would experience tinnitus or “ringing in the ears”.

4.3 Noise Dose Distribution

This section looks at the distribution of noise experienced by those working in the dental clinics and in the orthopaedic cast clinic.

4.3.1 A Comparison of Noise Dose Distribution between Dental Clinics and Orthopaedic Cast Clinic.

The distribution of sound pressure levels for the average daily noise dose at each dental surgery and in the orthopaedic cast clinic are shown in Table 4.5. This table reveals that the noise level was 65 dBA or above for 31.14%, 55.33%, 53.52% and 72.62% of the time in dental clinics 1, 2, 3, and

the orthopaedics clinic respectively. It can also be seen that the noise level was 75 dBA or above for 12.31%, 19.43%, 19.03% and 25.72% of the time in dental clinics 1, 2, 3, and the orthopaedics cast clinic respectively.

Average Daily Noise Dose Sound Pressure Level (dBA)	Clinic			
	1	2	3	OPR
45-49.9	1.97	1.27	4.10	0.42
50-54.9	35.90	12.33	17.93	3.68
55-59.9	18.00	15.70	10.40	7.60
60-64.9	12.80	15.70	13.87	15.64
65-69.9	10.10	17.40	16.26	24.04
70-74.5	8.73	18.50	18.23	22.86
75-79.9	9.17	13.60	15.70	14.58
80-84.9	2.87	5.00	3.10	7.40
85-89.9	0.27	0.83	0.23	2.94
90-94.9	0.00	0.00	0.00	0.74
95-99.9	0.00	0.00	0.00	0.06

Table 4.5: Average Daily Noise Dose Sound Pressure Level Distribution. The table shows the percentage distribution (%) of sound pressure levels, in dBA, measured in the dental surgeries and the orthopaedic cast clinic room.

The distribution of sound pressure levels for the average daily noise dose in clinic 1 and the cast clinic are shown in Figure 4.4.

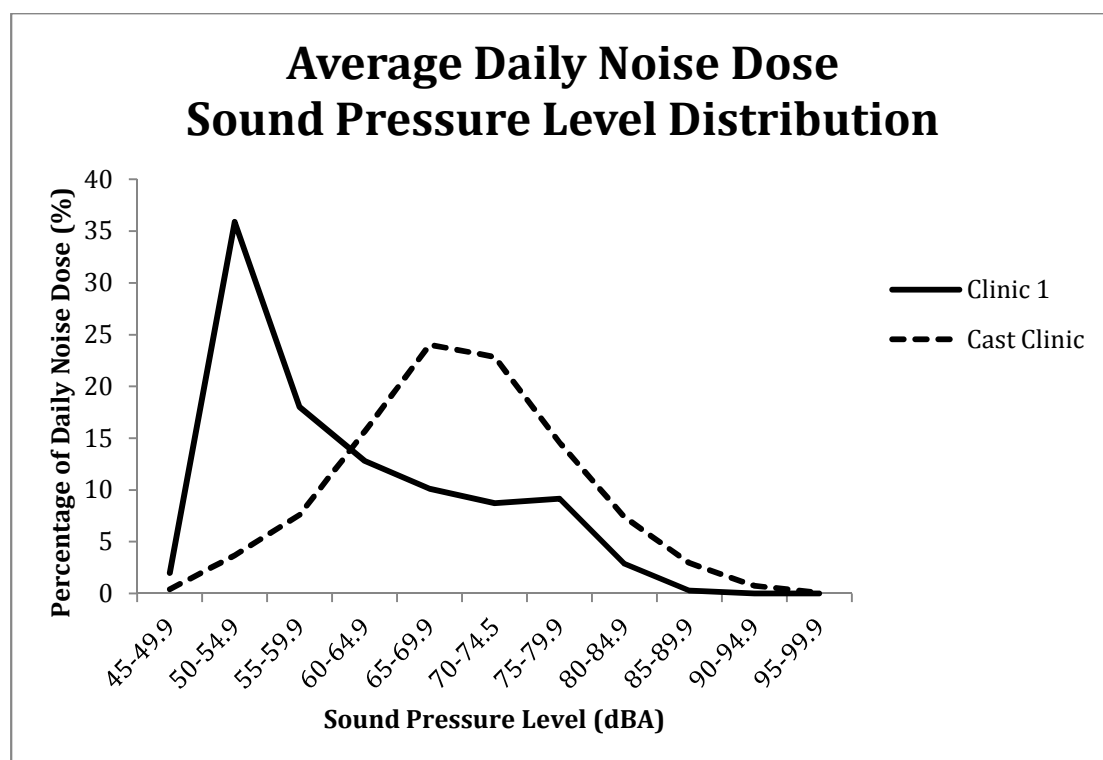


Figure 4.4: Average Daily Noise Dose Sound Pressure Level Distribution. The graph shows the percentage distribution (%) of sound pressure level in dBA for clinic 1 and the orthopaedic cast clinic.

5

Discussion

5.1 Summary

This chapter outlines the findings of the study and the possible consequences of these findings. Although based on a limited number of tests, the results are a starting point toward the need for further research into the auditory and non-auditory affects of noise in the New Zealand health industry.

5.2 Noise levels in the Health Industry

During Stage One of the study, when speaking to the dental staff at participating dental clinics, and some of their colleagues from other dental clinics, their concern about the noise levels became apparent. Dental staff perceive the noise levels in the industry to be high and the primary cause of hearing issues. However, the results from this study are similar to those reported by Sorainen and Rytönen, which showed that noise levels in dental clinics (Sorainen & Rytönen, 2002).

Although these results would suggest that there is no threat to hearing thresholds, Messano and Petti, 2012, found in a questionnaire survey that dental staff were two times more likely to report experiencing hearing loss and two and a half times more likely to report experiencing tinnitus than age and work-experience matched medical staff (Messano & Petti, 2012). Messano and Petti's study however, did not include audiometric analysis and therefore conclusions could only be made on the dental staffs' perception of hearing dysfunction. If an actual hearing loss exists amongst dental staff it may, as reviewed elsewhere, be due to other factors, such as individual susceptibility, presbycusis and/or sociocusis, or other ototoxic factors present in the dental clinics, as described in Appendix 6.

The cause of the dental staffs' concern may result from the non-auditory health affects of noise rather than the auditory affects. Noise levels in the dental surgeries were recorded at 65 dBA or above for 31% to 53% of the working day. This level of noise, and probably also its tonal characteristics, would result in increased levels of annoyance and stress effecting concentration levels and decision-making abilities. Ultimately this could

result in decreased productivity and possibly an increased risk of errors in judgment and fine precision work.

In the health industry, staff-to-staff and staff-to-patient/client communication is vital. Webster, 1979, recommends that the A-weighted sound level, in work spaces where speech communication is essential, not exceed 62 dBA so as to not reduce speech intelligibility or cause communication interference. In dentistry although the noise levels may exceed 62 dBA at times, the clinician has control over when noise is produced, in that, if they wish to speak to the client/patient they can stop drilling thus reducing communication interference.

Environmental noise levels of 55-65 dBA have been linked to increased levels of the stress hormones cortisol, adrenaline and noradrenalin, which result in adverse health effects, such as, changes in the Ca/Mg concentrations in myocardium. With an L_{eq8h} of 70 - 73 dBA, staff working in dental clinics are at risk of such non-auditory health affects. Dental professionals in private practice are often owner operators and generally do not have a hearing conservation or health monitoring programmes in place.

During the second stage of the study when talking with the orthopaedic staff, their concern about the noise levels was also evident. Whilst their perception was that noise levels were high, it was the non-auditory affects of the noise that appeared to be of more concern to them. They spoke of the high noise levels and the stress that created, and which at times forced them to leave the cast clinic. They spoke also of episodes of tinnitus related to high noise levels.

Orthopaedic staff were concerned about the threat to patient confidentiality. To communicate information effectively with other staff

members or patients/clients staff need to raise their voice above the background noise, which creates more noise. This is known as the Lombard effect, where noise creates more noise.

The results from this study, of an L_{eq8h} of 73-80 dBA, are similar to those found by Marsh and colleagues, 2011, which showed that noise levels in orthopaedic cast clinics do not exceed the maximum levels set out in Regulation 11 of the Health and Safety Act of L_{eq8h} of 85 dBA (Marsh et al., 2011). However, the workload in the cast clinic is dependent on the number of acute patients seen and the type and number of arranged clinics conducted on any one day in the Orthopaedic Outpatients Department. The days on which noise levels were measured were reported to be “quiet days” by staff. Although described as a “quiet day”, noise levels on day 2 were 27% of a maximum allowable daily noise dose, well above the 9% - 13% recorded on the remaining days. This increase in noise level was due to one paediatric patient screaming throughout the removal of their cast. Although it is not possible to predict the noise levels that may be recorded on a “noisy day”, it would be interesting to measure them.

As in the case of those working in the dental clinics, noise levels, and possibly the tonal characteristics of the noise, result in increased levels of annoyance and stress effecting cognitive skills, such as, concentration levels and decision making abilities. This is likely to lead to decreased productivity, increased absenteeism and, potentially, an increased risk of accidents, errors in judgment, and difficulties with fine precision work.

The noise levels in the cast clinic are high enough to cause interference with communication by reducing speech intelligibility. However unlike dentistry, where dental staff have some control over when noise is produced,

orthopaedic staff only have partial control. With up to eight clients plus their support network, medical teams, 7 cast clinic staff and up to 3 saws in operation at any one time, an individual staff member has little control over the background noise level. As reported by Job, 1996, the lack of control over the level of noise adds to the stress felt by staff. Orthopaedic staff are at greater risk of non-auditory health affects due to workplace noise exposure.

Christchurch Public Hospital does monitor the hearing of its orthopaedic staff as part of a hearing conservation programme but this programme does not monitor the non-auditory affects of noise exposure. Personal hearing protectors are issued to staff in the cast clinic but not all staff wear the earmuffs or earplugs provided as they find them cumbersome and make communication with staff and patients/clients even more difficult.

After reviewing the results of this research I would recommend that as well as routine hearing tests, the general health of staff working in the health industry be monitored on a regular basis. I would also recommend that if a hearing conservation programme is not already in existence that one be set up to inform staff of the affects on hearing health and general health of workplace noise, how to avoid the risk of hazardous noise exposure, and the importance of the use of personal hearing protection.

I would also recommend that a schedule for regular maintenance of equipment be set up to ensure that noise level dental and orthopaedic equipment is minimized and consideration be given to noise emission levels when purchasing new or replacement equipment.

5.3 References

- Marsh, J. P., Jellicoe, P., Black, B., Monson, R. C., & Clark, T. A. (2011). Noise levels in adult and pediatric cast clinics. *The American Journal of Orthopedics*, 40(7), E122-E124.
- Messano, G. A., & Petti, S. (2012). General dental practitioners and hearing impairment. *Journal of Dentistry*, 40, 821-828.
- Sorainen, E., & Rytönen, E. (2002). Noise level and ultrasound spectra during burring. *Clinical Oral Investigation*, 6, 133-136.

6

Conclusion and Future Research

6.1 Conclusion

In summary, this study highlighted some important points related to the noise levels in the health industry, in particular, dental surgeries and orthopaedic cast clinic. Firstly, it can be concluded that the noise levels in dental clinics unlikely to result in NIHL but could however, result in non-auditory effects for those working in the dental surgery environment. Secondly, it can be concluded that, although the noise levels in the orthopaedic cast clinic during a busy clinic may be high, overall they are unlikely to result in NIHL but could however, result in non-auditory effects for those working within that environment.

6.2 Future Research

This study has raised a few questions about noise safety in the health industry in New Zealand. Outlined below are some recommendations for further research in this field.

6.2.1 Noise Levels in New Zealand Orthopaedic Cast Clinics

The current study measured the noise levels at the Christchurch Public Hospital over a period of 5 working days; these days were considered by staff to be “quiet days.” Future research could involve:

1. Measurement of noise levels in cast clinics of other New Zealand Health Boards.
2. Measurements of noise levels in cast clinics recorded over a longer period so as to sample “busy days” as well as “quiet days.”
3. Measurement of the room acoustics in cast clinics and options for improvement of the acoustic working environments.

6.2.2 Noise Levels in New Zealand Dental Surgeries

The current study measured the room acoustics and noise levels in 3 private Christchurch dental surgeries. A more comprehensive study could be undertaken in the future to look at:

1. Noise levels in a larger sample of private and public dental surgeries and assessment of room acoustics.
2. Compare the noise levels produced by new and used dental equipment.
3. Narrow band analysis of noise emitted by various dental equipment and identification of noise sources.

6.2.3 Non-auditory Health Effects of Noise Levels in the Health Industry

Future research could include questionnaire surveys of health workers and the perceived effect of noise levels within the workplace on:

1. Noise annoyance
2. Work performance
3. Cognitive performance
4. Stress levels and,
5. General health and wellbeing

6.2.4 Stress-hormone Levels in Health Workers Subjected to Noise in the Workplace

Research monitoring the blood or urine levels of the stress hormones (cortisol, adrenaline and noradrenaline) of workers in the health industry who are subjected to continuous noise levels greater than L_{eq8h} 60 dBA.

A.1

Appendix 1 - Ambient Noise Level Raw Data

A.1.1 Summary

This section contains a description of each dental clinic and the results obtained during measurement of ambient noise levels using the procedure described in Chapter 3.

A.1.2 Ambient Noise Levels: Dental Clinics

All the ambient noise samples were made using a B&K 2250 during the clinicians' lunch break with no staff or patients present in the room.

Clinic 1 is a small clinic situated on the ground floor at the rear of the building so therefore is away from traffic noise. Traffic noise is minimal as the building is located in a quiet street. There is an air conditioning unit within the room and the room is adjacent to the preparation/sterilizing room. At the time of sampling there was a lot of activity in the preparation room and the air conditioning unit was operating.

Clinic 2 is a large clinic situated on the first floor at the front of the building and is exposed to traffic noise from a busy main road. The only window in the room faces the road. The window is double-glazed. At the time of sampling the air conditioning unit was operating.

Clinic 3 is a large clinic situated on the ground floor at the rear of the building so therefore is away from traffic noise. The building is located on a busy street. There is carpet on the floor in the clinic that covers about half of the floor space. At the time of sampling the air conditioning unit was operating. Only two samples were taken at this clinic, as the room was required for the treatment of patients.

The dental clinic ambient noise level raw data is shown below in Table A.1.1.

Sample	Dental Clinic		
	Clinic 1	Clinic 2	Clinic 3
Sample 1 (dBA)	40	39	37
Sample 2 (dBA)	40	39	35
Sample 3 (dBA)	40	39	-
Average (dBA)	40	39	36

Table A.1.1: Ambient Noise Level Raw Data. Results of measurements of ambient noise levels in dental clinics in dBA.

A.2

Daily Noise Dose Raw Data

A.2.1 Summary

The daily noise dose data present in the section was obtained with the B&K 4436 dosimeter using the methods described in Chapter 3.

A.2.2 Daily Noise Dose: Dental clinics

The tables in subsections A.2.2.1, A.2.2.2 and A.2.2.3 contain the daily noise dose data measured in the Clinics 1, 2 and 3 respectively.

A.2.2.1 Noise Dose Measurements: Clinic 1

	Noise Dose Measurements			
	Day 1	Day 2	Day 3	Average
Dose	4%	3%	13%	6.7%
Dose 8 hrs	6%	3%	12%	7.0%
Sound exposure	0.04 Pa ² h	0.03 Pa ² h	0.13 Pa ² h	0.06 Pa ² h
Sound exp. 8 hrs	0.06 Pa ² h	0.03 Pa ² h	0.12 Pa ² h	0.07 Pa ² h
LEP d	72.8	69.2	75.7	72.6
PSEL	71.1	69.2	75.8	72.0
Leq	72.8	69.2	75.7	72.6
Max L	103.2	105.8	128.4	112.5
Max P	139.9	141.8*	139.1	140.3
SEL	115.7	113.8	120.4	116.6

Table A.2.1 Daily Noise Dose Raw Data: Clinic 1. Daily dosimeter measurements recorded in Clinic 1. Noise dose (dose) and eight-hour equivalent noise dose (dose 8-hrs) are recorded as a percentage. Sound exposure and equivalent 8-hour sound exposure (sound exp. 8 hrs) recorded in Pa²h. LEP d, PSEL, Leq, Max l, Max P and SEL recorded in dBA.

Sound pressure level (dBA)	Percentage of daily dose (%)			
	Day 1	Day 2	Day 3	Average
45-49.9	4.9	0.8	0.2	1.97
50-54.9	23.8	53.5	30.4	35.90
55-59.9	14.6	15.2	24.2	18.00
60-64.9	15.0	8.6	14.8	12.80
65-69.9	12.4	7.0	10.9	10.10
70-74.5	9.2	6.7	10.3	8.73
75-79.9	13.4	6.3	7.8	9.17
80-84.9	6.0	1.8	0.8	2.87
85-89.9	0.4	0.0	0.4	0.27
90-94.9	0.0	0.0	0.0	0.00
95-99.9	0.0	0.0	0.0	0.00
100-104.9	0.0	0.0	0.0	0.00
105-109.9	0.0	0.0	0.0	0.00

Table A.2.2 Daily Noise Dose Distribution: Clinic 1. The distribution of the noise dose in clinic 2, in 5 dB increments, over the range from 45 dBA to 109.9 dBA.

A.2.2.2 Noise Dose Measurements: Clinic 2

Noise Dose Measurements				
	Day 1	Day 2	Day 3	Average
Dose	3%	4%	6%	4.3%
Dose 8 hrs	5%	4%	5%	4.7%
Sound exposure	0.03 Pa ² h	0.04 Pa ² h	0.06 Pa ² h	0.043 Pa ² h
Sound exp. 8 hrs	0.05 Pa ² h	0.04 Pa ² h	0.05 Pa ² h	0.047 Pa ² h
LEP d	72.3	70.5	72.2	71.7
PSEL	69.3	70.7	72.3	70.8
Leq	72.3	70.5	72.2	71.7
Max L	98.5	98.8	100.7	99.3
Max P	141.5	133.4	139.7	138.2
SEL	113.6	115.3	116.9	115.3

Table A.2.3 Daily Noise Dose Raw Data: Clinic 2. Daily dosimeter measurements recorded in Clinic 2. Noise dose (dose) and eight-hour equivalent noise dose (dose 8-hrs) are recorded as a percentage. Sound exposure and equivalent 8-hour sound exposure (sound exp. 8 hrs) recorded in Pa²h. LEP d, PSEL, Leq, Max l, Max P and SEL recorded in dBA.

Percentage of daily dose (%)				
Sound pressure level(dBA)	Day 1	Day 2	Day 3	Average
45-49.9	0.1	0.8	1.5	1.27
50-54.9	6.7	16.3	14.0	12.33
55-59.9	15.1	17.7	14.3	15.70
60-64.9	16.6	16.0	14.5	15.70
65-69.9	18.0	17.6	16.6	17.40
70-74.5	20.9	16.6	18.0	18.50
75-79.9	15.2	10.8	14.8	13.60
80-84.9	6.2	3.4	5.4	5.00
85-89.9	0.9	0.7	0.9	0.83
90-94.9	0.0	0.0	0.0	0.00
95-99.9	0.0	0.0	0.0	0.00
100-104.9	0.0	0.0	0.0	0.00
105-109.9	0.0	0.0	0.0	0.00

Table A.2.4 Daily Noise Dose Distribution: Clinic 2. The distribution of the noise dose in clinic 2, in 5 dB increments, over the range from 45 dBA to 109.9 dBA.

A.2.2.3 Noise Dose Measurements: Clinic 3

Noise Dose Measurements				
	Day 1	Day 2	Day 3	Average
Dose	3%	4%	4%	3.7%
Dose 8 hrs	3%	4%	4%	3.7%
Sound exposure	0.04 Pa ² h	0.04 Pa ² h	0.04 Pa ² h	0.04 Pa ² h
Sound exp. 8 hrs	0.03 Pa ² h	0.04 Pa ² h	0.04 Pa ² h	0.037 Pa ² h
LEP d	70.2	70.9	70.8	70.6
PSEL	70.3	71.3	70.8	70.8
Leq	70.2	70.9	70.8	70.6
Max L	99.9	96.0	101.9	99.3
Max P	136.1	131.9	133.4	133.8
SEL	114.9	115.9	115.4	115.3

Table A.2.5 Daily Noise Dose Raw Data: Clinic 3. Daily dosimeter measurements recorded in Clinic 3. Noise dose (dose) and eight-hour equivalent noise dose (dose 8-hrs) are recorded as a percentage. Sound exposure and equivalent 8-hour sound exposure (sound exp. 8 hrs) recorded in Pa²h. LEP d, PSEL, Leq, Max l, Max P and SEL recorded in dBA.

Percentage of daily dose (%)				
Sound pressure level(dBA)	Day 1	Day 2	Day 3	Average
45-49.9	5.1	1.8	5.4	4.10
50-54.9	15.2	17.4	21.2	17.93
55-59.9	8.0	9.2	14.0	10.40
60-64.9	13.0	14.7	13.9	13.87
65-69.9	18.6	16.5	13.7	16.26
70-74.5	22.1	19.0	13.6	18.23
75-79.9	14.9	17.7	14.5	15.70
80-84.9	2.6	3.0	3.7	3.10
85-89.9	0.2	0.3	0.2	0.23
90-94.9	0.0	0.0	0.0	0.00
95-99.9	0.0	0.0	0.0	0.00
100-104.9	0.0	0.0	0.0	0.00
105-109.9	0.0	0.0	0.0	0.00

Table A.2.6 Daily Noise Dose Distribution: Clinic 3. The distribution of the noise dose in clinic 3, in 5 dB increments, over the range from 45 dBA to 109.9 dBA.

A.2.2.4 Noise Dose Measurements: Orthopaedic Cast Clinic

Table A.2.7, A.2.8 contain the daily noise dose and distribution data measured in the Orthopaedic cast clinic.

Noise Dose Measurements						
	Day 1	Day 2	Day 3	Day 4	Day 5	Average
Dose (%)	5	21	9	11	13	11.8
Dose 8 hr (%)	6	27	9	11	13	13.2
Sound exposure (Pa²h)	0.05	0.21	0.09	0.11	0.13	0.12
Sound exp. 8 hrs (Pa²h)	0.06	0.27	0.09	0.11	0.14	0.13
LEP d	72.9	79.3	74.5	75.4	76.2	75.7
PSEL	72.2	78.0	74.2	75.3	76.0	75.1
Leq	72.9	79.3	74.5	75.4	76.2	75.7
Max L	100.7	104.7	99.5	105.6	113.4	104.8
Max P	139.0	132.9	138.1	135.2	134.9	136.0
SEL	116.0	122.6	118.8	119.9	120.6	119.6

Table A.2.7 Daily Noise Dose Raw Data: Orthopaedic Cast Clinic. Daily dosimeter measurements recorded in the orthopaedic cast clinic. Noise dose (dose) and eight-hour equivalent noise dose (dose 8-hrs) are recorded as a percentage. Sound exposure and equivalent 8-hour sound exposure (sound exp. 8 hrs) recorded in Pa²h. LEP d, PSEL, Leq, Max L, Max P and SEL recorded in dBA.

Percentage of daily dose (%)						
Sound pressure level (dBA)	Day 1	Day 2	Day 3	Day 4	Day 5	Average
45-49.9	0.1	0.2	0.4	0.5	0.9	0.4
50-54.9	3.4	2.7	3.0	5.1	4.2	3.7
55-59.9	7.6	5.5	7.0	8.9	9.0	6.0
60-64.9	17.1	11.6	15.1	16.9	17.5	15.6
65-69.9	26.6	18.7	25.1	23.3	26.5	24.0
70-74.5	25.2	22.9	24.5	20.5	21.2	22.9
75-79.9	13.1	18.7	15.0	13.3	12.8	14.6
80-84.9	5.0	11.4	7.0	7.9	5.7	7.4
85-89.9	1.5	6.0	2.3	3.1	1.8	2.9
90-94.9	0.2	2.1	0.5	0.6	0.3	0.7
95-99.9	0.0	0.3	0.0	0.0	0.0	0.1
100-104.9	0.0	0.0	0.0	0.0	0.0	0.0
105-109.9	0.0	0.0	0.0	0.0	0.0	0.0

Table A.2.8 Daily Noise Dose Distribution: Orthopaedic Cast clinic. The distribution of the noise dose in the orthopaedic cast clinic, in 5 dB increments, over the range from 45 dBA to 109.9 dBA.

A.3

Reverberation Time Raw Data

A.3.1 Summary

Reverberation times were measured in each dental clinic and background noise levels were measured in the orthopaedic cast clinic using a sound level meter using the methods described in Chapter 3.

A.3.2 Reverberation Times in Dental clinics

Reverberation time measurements were taken, using the methods as described in Chapter 3, with the B&K 2250 sound analyzer.

A.3.2.1 Reverberation Time: Clinic 1

RT20				RT30		
Frequency (Hz)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int. (s)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int.(s)
100	0.415	0.062	0.061	0.413	0.030	0.029
125	0.308	0.054	0.053	0.335	0.037	0.036
160	0.268	0.033	0.032	0.280	0.008	0.008
200	0.275	0.067	0.065	0.310	0.061	0.059
250	0.308	0.013	0.012	0.315	0.013	0.013
315	0.353	0.095	0.093	0.328	0.057	0.056
400	0.363	0.026	0.026	0.365	0.045	0.044
500	0.370	0.100	0.098	0.373	0.049	0.048
630	0.380	0.042	0.042	0.373	0.022	0.022
800	0.408	0.029	0.028	0.395	0.037	0.036
1000	0.370	0.024	0.024	0.388	0.021	0.020
1250	0.425	0.041	0.040	0.413	0.039	0.039
1600	0.408	0.039	0.038	0.413	0.017	0.017
2000	0.440	0.018	0.018	0.433	0.017	0.017
2500	0.428	0.033	0.032	0.438	0.022	0.022
3150	0.438	0.025	0.024	0.445	0.013	0.013
4000	0.443	0.017	0.017	0.458	0.013	0.012
5000	0.465	0.010	0.010	0.470	0.016	0.016

Table A.3.1. Reverberation Time Raw Data: Clinic 1. Reverberation times (RT60), in seconds (s), extrapolated from measurements of RT20 and RT30 with standard deviation (std. dev.) and 95% confidence interval (95% confidence int.) measured in seconds (s).

A.3.2.2 Reverberation Time: Clinic 2

RT20				RT30		
Frequency (Hz)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int.(s)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int.(s)
100	1.36	0.505	0.495	1.36	0.505	0.495
125	0.52	0.103	0.101	1.48	1.626	1.593
160	0.58	0.127	0.124	0.60	0.084	0.082
200	0.65	0.114	0.111	0.58	0.045	0.044
250	0.60	0.043	0.043	0.62	0.031	0.030
315	0.70	0.106	0.104	0.69	0.032	0.031
400	0.81	0.130	0.127	0.81	0.105	0.103
500	0.70	0.068	0.066	0.73	0.046	0.045
630	0.77	0.058	0.057	0.78	0.057	0.056
800	0.77	0.039	0.038	0.81	0.021	0.020
1000	0.91	0.061	0.059	0.90	0.034	0.033
1250	0.98	0.068	0.066	0.97	0.066	0.065
1600	0.98	0.019	0.019	1.00	0.025	0.024
2000	0.95	0.029	0.028	0.98	0.022	0.022
2500	0.87	0.070	0.068	0.89	0.039	0.039
3150	0.86	0.013	0.013	0.90	0.013	0.013
4000	0.92	0.029	0.028	0.93	0.013	0.013
5000	0.86	0.029	0.028	0.85	0.021	0.020

Table A.3.2. Reverberation Time Raw Data: Clinic 2. Reverberation times (RT60), in seconds (s), extrapolated from measurements of RT20 and RT30 with standard deviation (std. dev.) and 95% confidence interval (95% confidence int.) measured in seconds (s).

A.3.2.3 Reverberation Time - Clinic 3

RT20				RT30		
Frequency (Hz)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int.(s)	Average RT60 (s)	Std. Dev. (s)	95% Confidence Int.(s)
100	0.29	0.026	0.026	0.29	0.026	0.026
125	0.36	0.042	0.041	0.69	0.648	0.635
160	0.41	0.014	0.014	0.41	0.059	0.058
200	0.46	0.139	0.136	0.42	0.086	0.084
250	0.51	0.036	0.035	0.52	0.033	0.033
315	0.47	0.110	0.108	0.44	0.096	0.094
400	0.49	0.026	0.026	0.47	0.022	0.022
500	0.35	0.075	0.073	0.36	0.026	0.026
630	0.39	0.024	0.023	0.37	0.006	0.006
800	0.37	0.065	0.064	0.39	0.042	0.042
1000	0.40	0.054	0.053	0.38	0.021	0.020
1250	0.38	0.021	0.020	0.37	0.015	0.015
1600	0.39	0.041	0.040	0.40	0.013	0.012
2000	0.38	0.022	0.022	0.38	0.005	0.005
2500	0.39	0.037	0.036	0.40	0.017	0.017
3150	0.37	0.010	0.009	0.37	0.018	0.018
4000	0.38	0.024	0.023	0.38	0.006	0.006
5000	0.39	0.013	0.012	0.38	0.010	0.010

Table A.3.3. Reverberation Time Raw Data: Clinic 3. Reverberation times (RT60), in seconds (s), extrapolated from measurements of RT20 and RT30 with standard deviation (std. dev.) and 95% confidence interval (95% confidence int.) measured in seconds (s).

A.4

Appendix 4 – Spectral Analysis Raw Data

A.4.1 Summary

This section contains measurements of the spectral content of the noise emitted from equipment used in each dental clinic.

A.4.2 Spectral Analysis of Dental Equipment

The measurements taken using a B&K 2250 spectral analyzer at clinics 1, 2 and 3 can be seen in Figures A.4.1, A.4.2 and A.4.3 respectively. It can be seen in all three graphs that the sound energy rises steadily above 400 Hz. A high sound pressure level at these frequencies between 400 Hz and 8 kHz will have the greatest impact on the ability to hear speech clearly (Sydney et al., 2007). Discussion on the following results can be found in Chapter 4.

Spectral Analysis: Clinic 1

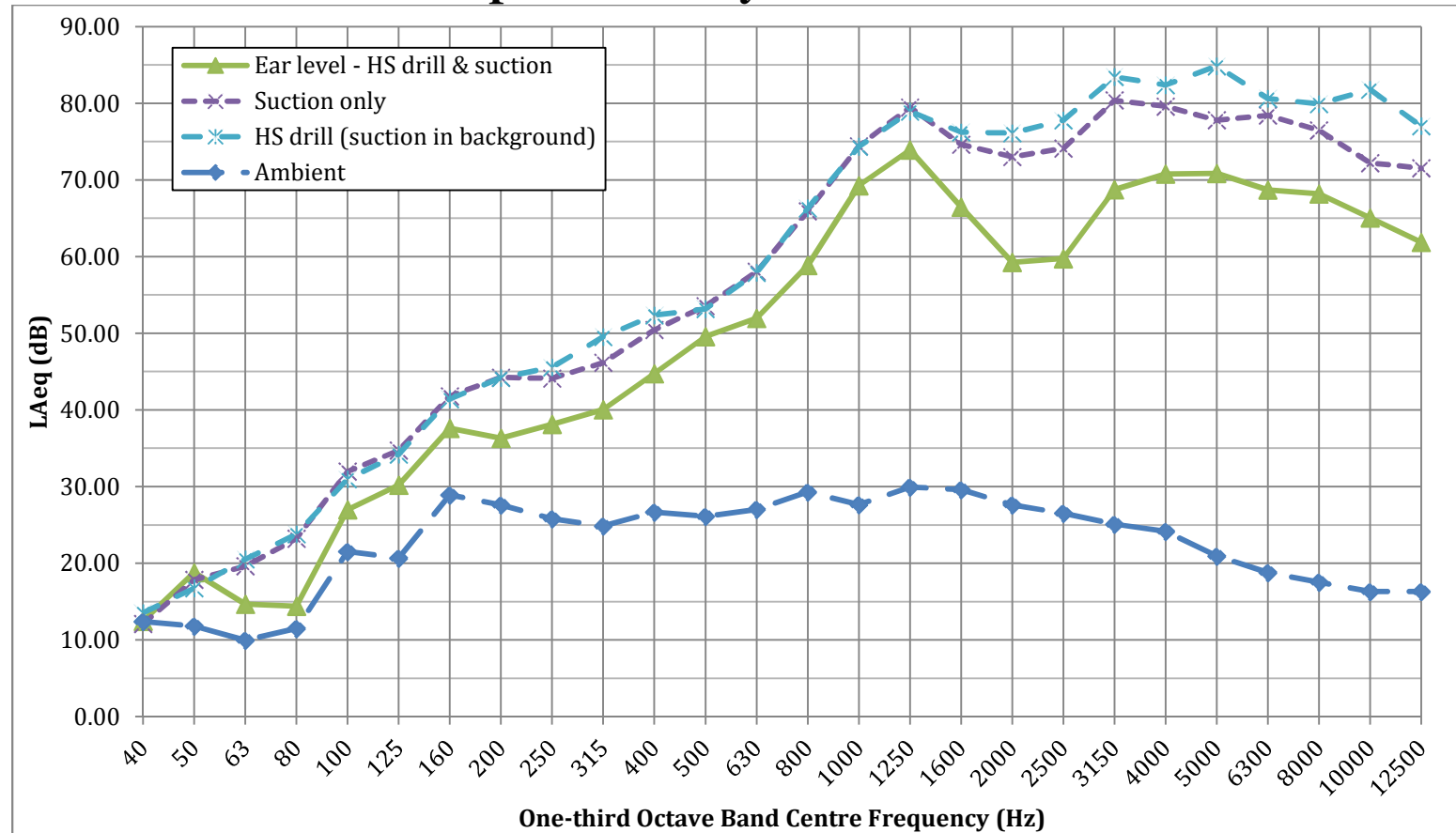


Figure A.4.1 Noise Spectrum: Clinic 1. The A-weighted equivalent level (LAeq) in dB at the one-third octave band frequency, in Hz, for the noise emitted by dental equipment. **Ear level – HS drill & suction:** spectral analysis of noise at ear level with the high speed drill and the suction in operation. **Suction only:** spectral analysis of noise within 2cm of the suction in operation. **HS drill (suction in background):** spectral analysis of noise within 2cm of the high speed drill in operation with the suction operating in the background. **Ambient:** spectral analysis of the ambient noise while the clinic was not in use.

Spectral Analysis: Clinic 2

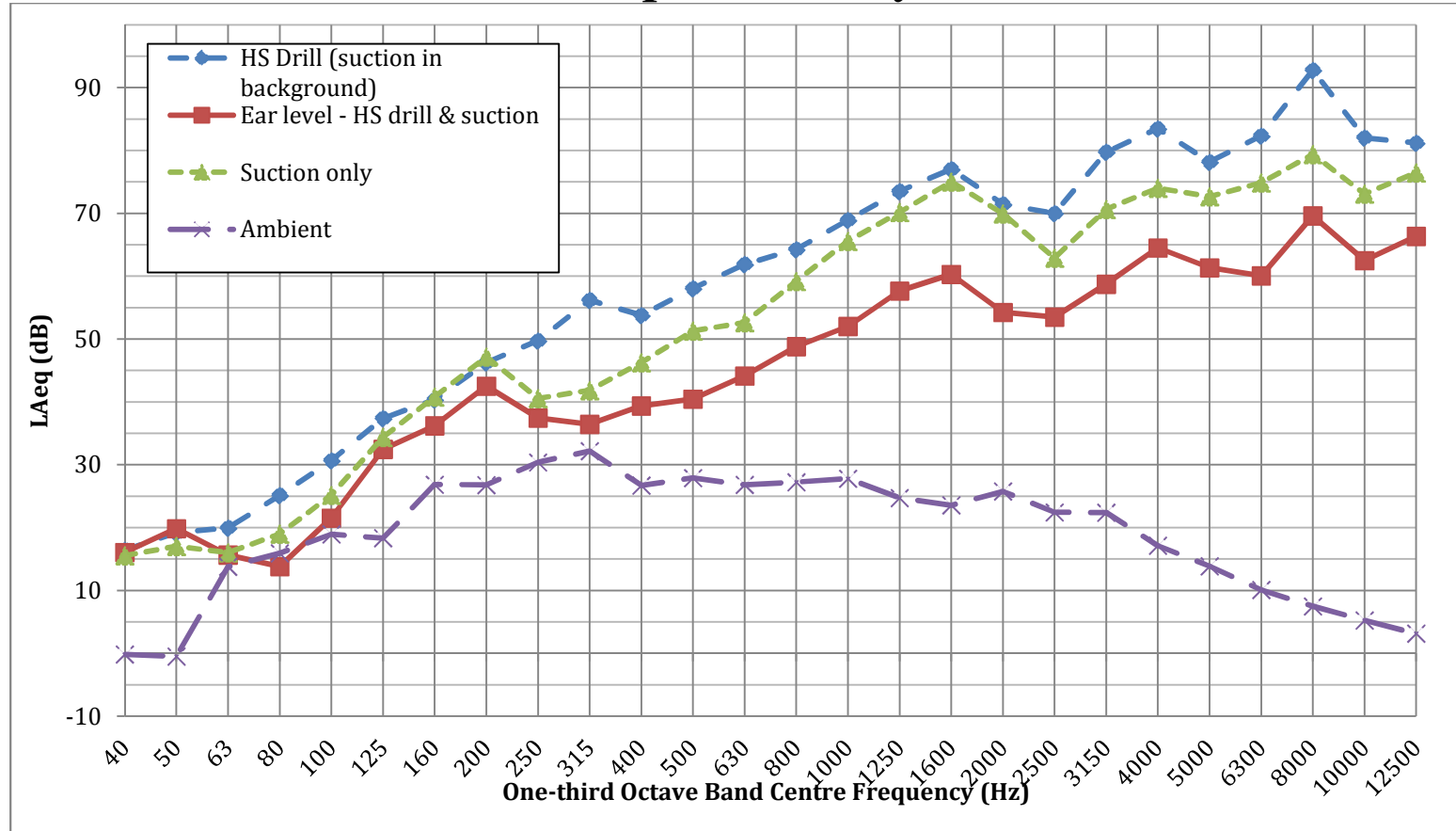


Figure A.4.2 Noise Spectrum: Clinic 2. The A-weighted equivalent level (LAeq) in dB at the one-third octave band frequency, in Hz, for the noise emitted by dental equipment. **Ear level – HS drill & suction:** spectral analysis of noise at ear level with the high speed drill and the suction in operation. **HS drill (suction in background):** spectral analysis of noise within 2cm of the high speed drill in operation with the suction operating in the background. **Suction only:** spectral analysis of noise within 2cm of the suction in operation. **Ambient:** spectral analysis of the ambient noise while the clinic was not in use.

Spectral Analysis: Clinic 3

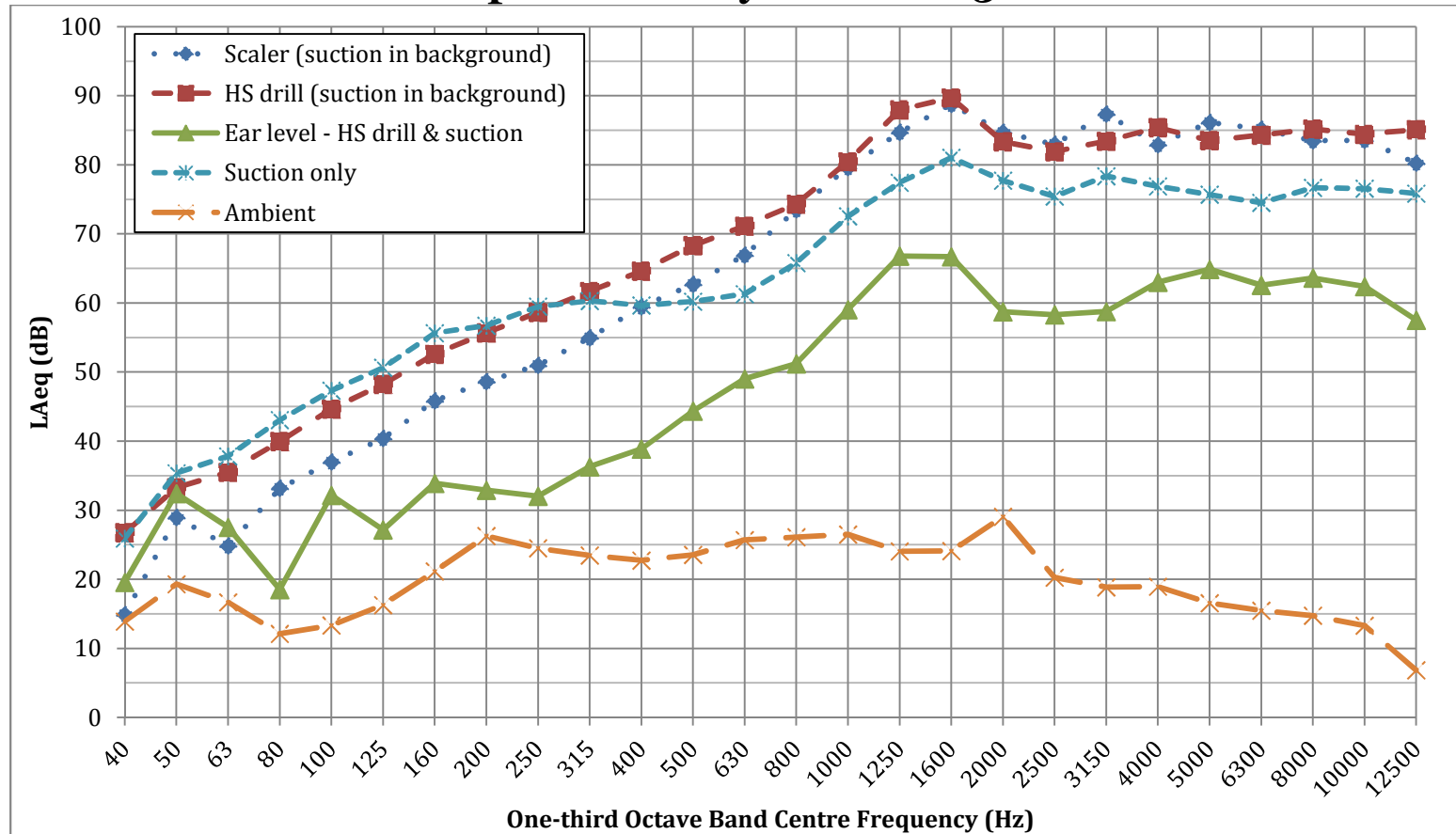


Figure A.4.3 Noise Spectrum: Clinic 3. The A-weighted equivalent level (LAeq) in dB at the one-third octave band frequency, in Hz, for the noise emitted by dental equipment. **Scaler:** spectral analysis of noise within 2 cm of the scaler in operation with the suction operating in the background. **HS drill (suction in background):** spectral analysis of noise within 2cm of the high speed drill in operation with the suction operating in the background. **Ear level – HS drill & suction:** spectral analysis of noise at ear level with the high speed drill and the suction in operation. **Suction only:** spectral analysis of noise within 2cm of the suction in operation. **Ambient:** spectral analysis of the ambient noise while the clinic was not in use.

A.4.3 References

Sydney, S. E., Lepp, A. J., Whitehouse, S. L., & Crawford, R. W. (2007). Noise exposure due to orthopedic saws in simulated total knee arthroplasty surgery. *The Journal of Arthroplasty*, 22(8), 1193-1197.

A.5

Appendix 5 – Sabine Calculations Raw Data

A.5.1 Summary

This section contains Sabine calculations using room measurements and absorbency characteristics of the main fittings and fixtures for each dental clinic.

A.5.2 Sabine Calculations

Complex models using Sabine calculations are beyond the scope of this study; the models were simplified to include only the most important items common to all three dental clinics. Items included in the calculations were floor, wall and ceiling coverings along with windows, benches and the dental chair.

The absorption coefficients for the surface materials in the dental surgeries are shown in Table A.5.1 (Harris, 1991).

Sound Absorption Coefficients						
No.	125 Hz	250 Hz	500 Hz	1000 Hz	2000 Hz	4000 Hz
S1	0.29	0.10	0.06	0.05	0.04	0.04
S2	0.04	0.04	0.07	0.06	0.06	0.07
S3	0.02	0.03	0.03	0.03	0.03	0.02
S4	0.72	0.79	0.83	0.84	0.83	0.79
S5	0.35	0.25	0.18	0.12	0.07	0.04
S6	0.01	0.02	0.06	0.15	0.25	0.45

Table A.5.1 Sound Absorption Coefficients. Sound absorbcency coefficient for plasterboard (S1), wood (S2), vinyl (S3), a chair (S4), glass (S5) and carpet (S6).

A.5.2.1 Sabine Calculations: Clinic 1

The room area measurements used in the model for Clinic 1 can be seen below in Table A.5.2. An example of microphone and speaker placement, and general layout of Clinic 1 can be seen in figures A.5.1 and A.5.3. Figure A.5.2 shows the adjacent preparation room.

Room Areas			
No.	Material	Location	Area (m ²)
S1	Plasterboard	Walls and ceiling	27.1
S2	Wood	Cabinetry	15.9
S3	Vinyl	Flooring	11.4
S4	Chair		2.8
S6	Glass	Window	9.8
Total Area (m ²)			67.0
Room Volume (m ³)			29.37

Table A.5.2 Room Area Measurements: Clinic 1. This table includes surface area measurements, in square metres (m²), of the items common to all three dental clinics along and the total surface area of reflective surfaces, along with the room volume, in cubic metres (m³). S1-S6 refer to the sound absorbcency coefficients given in table A.5.1.



Figure A.5.1 Clinic 1. Photograph of Clinic 1 during reverberation time measurement with the speaker in position S1 and sound analyzer in position R1.

The following table, Table A.5.3, shows the results of the Sabine calculations using the room area measurements and the sound absorption coefficients from Tables A.5.2 and A.5.1 respectively. These results can be seen in Chapter 4 plotted against the measured reverberation time in Clinic 1.

ABSORPTION AREAS (m²)						
Frequency (Hz)	125	250	500	1000	2000	4000
S1	7.86	2.71	1.63	1.36	1.08	1.08
S2	0.64	0.64	1.11	0.95	0.95	1.11
S3	0.23	0.34	0.34	0.34	0.34	0.23
S4	2.02	2.21	2.32	2.35	2.32	2.21
S5	3.43	2.46	1.76	1.18	0.69	0.39
Results (s)	0.33	0.57	0.69	0.77	0.88	0.94

Table A.5.3 Calculated Reverberation Time: Clinic 1. Using the Sabine calculation the reverberation time is calculated from the sound absorption coefficients and the room area measurements from Table A.5.2.



Figure A.5.2 Clinic 1 and Adjacent Preparation Room.



Figure A.5.3 Clinic 1. Photograph of Clinic 1 during reverberation time measurement with the speaker in position S2 and sound analyzer in position R1.

A.5.2.2 Sabine Calculations: Clinic 2

The room area measurements used in the model for Clinic 2 can be seen below in Table A.5.4. An example of microphone and speaker placement, and general layout of Clinic 2 can be seen in Figure A.5.4.

Room Areas			
No.	Material	Location	Area (m ²)
S1	Plasterboard	Walls and ceiling	42.6
S2	Wood	Benches	7.6
S3	Vinyl	Flooring	13.9
S4	Chair		5.1
S6	Glass	Window	2.7
Total Area (m ²)			71.9
Room Volume (m ³)			35.62

Table A.5.4 Room Area Measurements: Clinic 2. This table includes surface area measurements, in square metres (m²), of the items common to all three dental clinics along and the total surface area of reflective surfaces, along with the room volume, in cubic metres (m³). S1-S6 refer to the sound absorbency coefficients given in table A.5.1.



Figure A.5.4 Clinic 2. Photograph of Clinic 2 during reverberation time measurement microphone in position R1.

The following table, Table A.5.5, shows the results of the Sabine calculations using the room area measurements and the sound absorption coefficients from Tables A.5.4 and A.5.1 respectively. These results can be seen in Chapter 4 plotted against the measured reverberation time in Clinic 2.

ABSORPTION AREAS (m²)						
Frequency (Hz)	125	250	500	1000	2000	4000
S1	12.35	4.26	2.56	2.13	1.70	1.70
S2	0.30	0.30	0.53	0.46	0.46	0.53
S3	0.28	0.42	0.42	0.42	0.42	0.28
S4	3.67	4.03	4.23	4.28	4.23	4.03
S5	0.95	0.68	0.49	0.32	0.19	0.11
Results (s)	0.33	0.59	0.70	0.75	0.82	0.86

Table A.5.5 Calculated Reverberation Time: Clinic 2. Using the Sabine calculation the reverberation time is calculated from the sound absorption coefficients and the room area measurements from Table A.5.4.

A.5.2.3 Sabine Calculations: Clinic 3

The room area measurements used in the model for Clinic 3 can be seen below in Table A.5.6. An example of microphone and speaker placement, and general layout of Clinic 3 can be seen in Figure A.5.5 and Figure A.5.6.

Room Areas			
No.	Material	Location	Area (m ²)
S1	Plasterboard	Walls and ceiling	36.6
S2	Wood	Benches	8.5
S3	Vinyl	Flooring	9.8
S4	Chair		6.2
S5	Glass	Window	3.1
S6	Carpet	Flooring	3.0
Total Area (m ²)			67.2
Room Volume (m ³)			29.98

Table A.5.6 Room Area Measurements: Clinic 3. This table includes surface area measurements, in square metres (m²), of the items common to all three dental clinics along and the total surface area of reflective surfaces, along with the room volume, in cubic metres (m³). S1-S6 refer to the sound absorbency coefficients given in table A.5.1.



Figure A.5.5 Clinic 3. Photograph of Clinic 3 during reverberation time measurement microphone in position R1 and the speaker in position S1.



Figure A.5.6 Clinic 3. Photograph of Clinic 3 during reverberation time measurement microphone in position R1 and the speaker position S2.

The following table, Table A.5.7, shows the results of the Sabine calculations using the room area measurements and the sound absorption coefficients from Tables A.5.6 and A.5.1 respectively. These results can be seen in Chapter 4 plotted against the measured reverberation time in Clinic 3.

ABSORPTION AREAS (m²)						
Frequency (Hz)	125	250	500	1000	2000	4000
S1	10.61	3.66	2.20	1.83	1.46	1.46
S2	0.34	0.34	0.60	0.51	0.51	0.60
S3	0.20	0.29	0.29	0.29	0.29	0.20
S4	4.46	4.90	5.15	5.21	5.15	4.90
S5	1.09	0.78	0.56	0.37	0.22	0.12
S6	0.03	0.06	0.18	0.45	0.75	1.35
Results (s)	0.29	0.48	0.54	0.56	0.58	0.56

Table A.5.7 Calculated Reverberation Time: Clinic 3. Using the Sabine calculation the reverberation time is calculated from the sound absorption coefficients and the room area measurements from Table A.5.6.

A.5.3 Clinic Layout

The layout of the Clinics 1, 2 and 3 can be seen in the following figures, Figures A.5.7, A.5.8 and A.5.9 respectively.

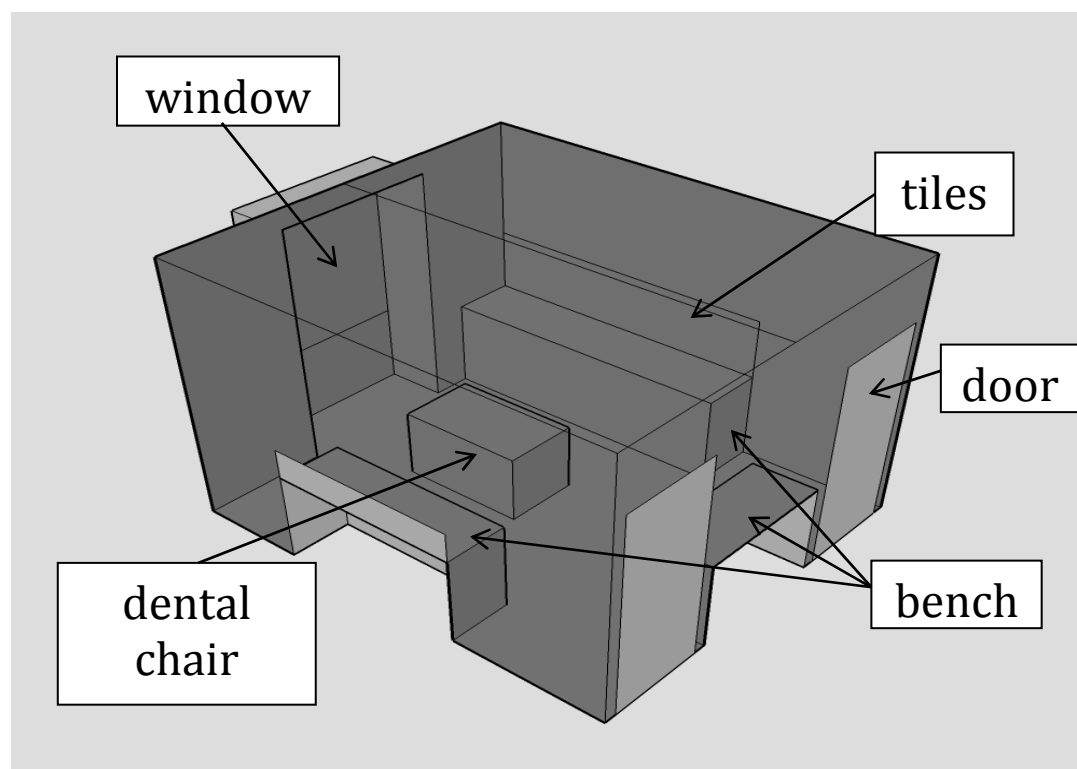


Figure A.5.7 Clinic 1 Layout

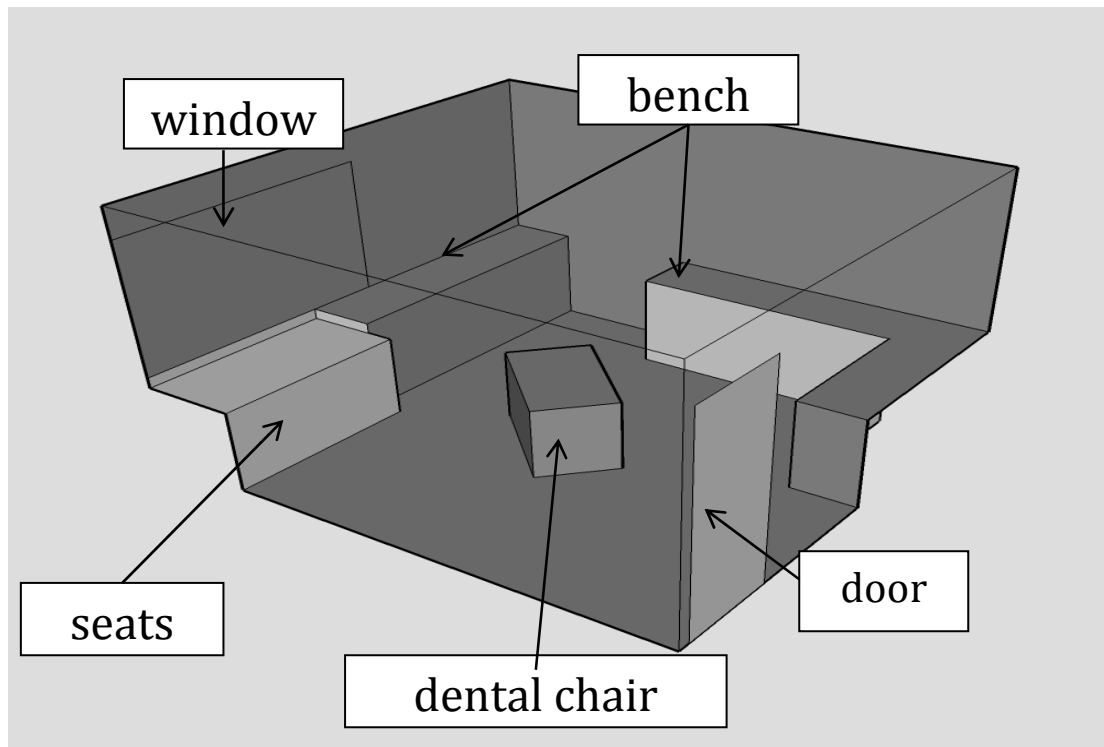


Figure A.5.8 Clinic 2 Layout

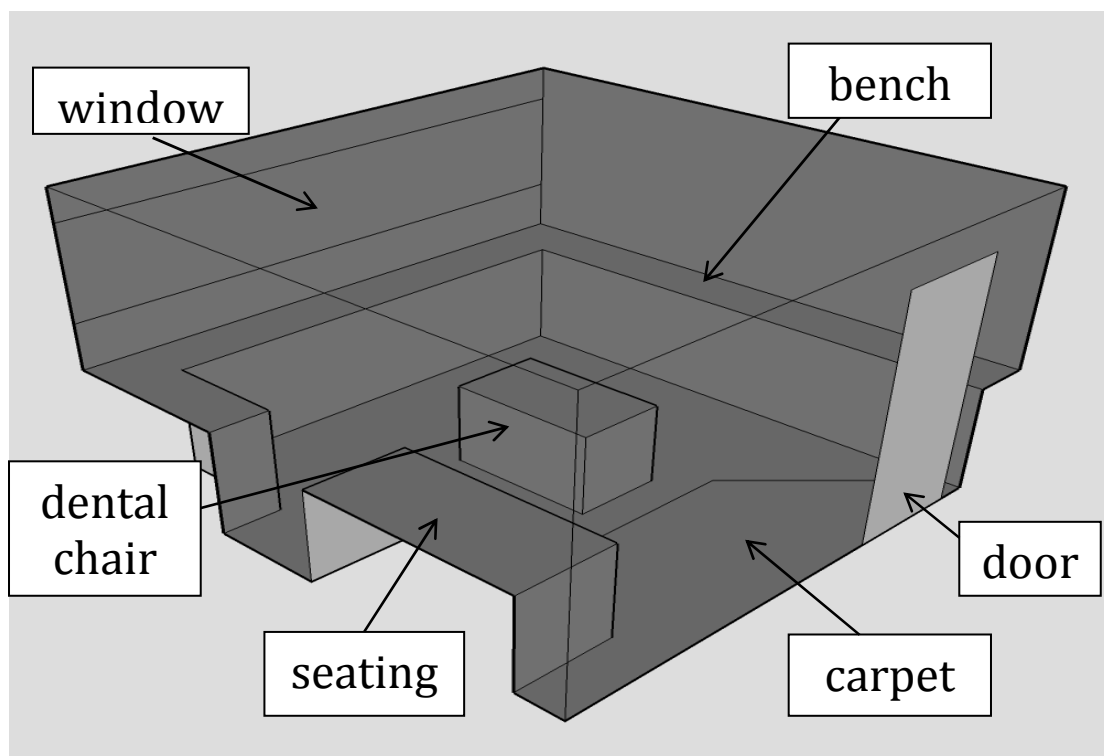


Figure A.5.9 Clinic 3 Layout

A.5.4 Discussion

The calculated reverberation times using the Sabine equation, which assumes that the sound absorption is distributed uniformly within the room and that the sound field is diffuse. The room used in the calculations is therefore an approximation of a real room and some variations between the measured and calculated reverberation times are expected. In these simple models the calculated results are higher than the measured RT60s; if a more detailed model had been used the calculated RT60s may have been closer to the measured RT60s. The calculated reverberation times for all three dental clinics can be seen in Table A.5.8 and the graphed along with the measured RT60s in Chapter 4.

Results (s)	Frequency (Hz)					
	125	250	500	1000	2000	4000
Clinic 1	0.35	0.57	0.66	0.77	0.88	0.94
Clinic 2	0.33	0.59	0.70	0.75	0.82	0.86
Clinic 3	0.29	0.48	0.54	0.56	0.58	0.56

Table A.5.8 Calculated Reverberation Times for Dental Clinics 1, 2 & 3.

The results for the calculations for Clinic 1 show the worst agreement with the measured reverberation times out of the three rooms. This is due, in part, to greater differences between the sound absorption coefficients used in the Sabine calculations and the true absorption properties of the real surfaces, in Clinic 1 than in the other two clinics. Clinic 1 also contained numerous smaller items, such as, plants and equipment, which provide extra sound absorption in the real room that were not included in the Sabine calculations.

The results from the Sabine equation for Clinics 2 and 3 show better agreement with the measured reverberation times and can therefore be used to give a good idea of the reverberation times that can be achieved if the treated in some way.

The measured reverberation times for Clinic 2 were the highest of the three clinics ranging from 0.52 to 1.36s in the low frequencies below 250 Hz, and 0.60s to 0.98s at frequencies between 250 Hz and 5000 Hz. These results fall outside the Australian/New Zealand Standard AS/NZS 2107:2000 recommended maximum reverberation time in medical rooms of 0.60s. A reduction in the reverberation time could be achieved if the room was to be treated in some way, for example, replacing some of the vinyl floor covering with carpet.

Using a modified model the room was treated with 6 m² of carpet in an attempt to lower the reverberation time of Clinic 2 to a more acceptable level. Table A.5.9 shows the results of the Sabine calculations for both Clinic 2 models, and reveals that the modification would lead to a reduction in the reverberation times above 500 Hz. As can be seen in Table A.5.1, carpet has relatively poor sound absorption properties at low frequencies.

	Frequency (Hz)					
Results (s)	125	250	500	1000	2000	4000
Vinyl	0.33	0.59	0.70	0.75	0.82	0.86
Vinyl/carpet	0.33	0.60	0.68	0.69	0.69	0.62

Table A.5.9 Calculated Reverberation Time: Clinic 3 with Modifications. Using the Sabine calculation the reverberation time is calculated from the sound absorption coefficients and the room area measurements from Table A.5.6.

A.5.5 References

Harris, C. M. (1991). *Handbook of Acoustical Measurements and Noise Control* (3rd ed.). New York: McGraw-Hill.

A.6

Appendix 6 - Additional Factors Affecting Hearing Loss

A.6.1 Summary

Appendix 6 identifies and reviews previous work on additional factors that may affect the hearing health of individuals working in the health industry.

A.6.2 Noise-induced hearing loss and chemicals

Many chemicals are known to have harmful effects on cochlear hair cells these include asphyxiates (carbon monoxide and hydrogen cyanide), some nitriles (such as acrylonitrile), and metals (lead, mercury and tin). Many therapeutic agents such as salicylates, non-steroidal anti-inflammatory drugs, loop-diuretics and the group of antibiotics, aminoglycosides are also known to cochlear hair cell damage. These therapeutic agents along with cytotoxic agents, such as cisplatin, are all reported to have ototoxic as well as nephrotoxic effects in humans(Rybak & Ramkumar, 2007). Other ototoxic agents have been have been shown in animal studies to have a synergistic effect along with noise causing decreased audiological thresholds. The role of all chemicals in human ototoxicity is still under evaluation but should be taken into consideration when evaluating sensorineural hearing loss (Kircher, 2003; Kircher et al., 2012).

A.6.2.1 Diuretics

Used commonly in the treatment of hypertension and oedema associated with congestive heart failure or renal insufficiency, diuretics increase the excretion of excess body fluids by means of a forced diuresis. Loop diuretics act primarily on the ascending loop of Henle in the kidneys resulting in changes to Na-K-2Cl transportation across cell membranes (Katsuhisa, Takeshi, Hiroshi, & Tomonori, 1997). These alterations to the Na-K-2Cl transportation also occur in the cochlea and are thought to be the common mechanism causing ototoxicity and nephrotoxicity (Hoffman, Whitworth, Jones, & Rybak, 1987; Katsuhisa et al., 1997).

The use of loop diuretics, such as Furosemide and Lasix, in high doses may cause a permanent sensorineural hearing loss (Stach, 1998). Post-mortem histological results show cystic and oedematous changes in the stria vascularis with little or no hair cell loss (Katsuhisa et al., 1997).

A.6.2.2 Painkillers

Many commonly used, across the counter pain killers, such as aspirin (acetylsalicylic acid), panadol (acetaminophen) and brufen or nurophen (ibuprofen), are known to have a negative effect on hearing thresholds (Canlon, Fransson, & Dagli, 1998; Curhan, Roland, Shargorodsky, & Curhan, 2010).

Consumption of high doses of ibuprofen and other non-steroidal anti-inflammatory drugs (NSAIDs) can result in reversible hearing loss and tinnitus (Davison & Marion, 1998; McKinnon & Lassen, 1998). The ototoxic effect of NSAIDs causes a reduction in blood flow to the cochlea resulting in reduced hearing thresholds (McFadden & Plattsmier, 1983; McFadden, Plattsmier, & Pasanen, 1984; Canlon et al., 1998; Curhan et al., 2010).

When taken in low doses salicylates have been found to offer some protection against the ototoxic effects of aminoglycoside antibiotics (Chen et al., 2007; Kimitsuki et al., 2009) and NIHL (Kopke et al., 2000). However when taken in higher doses, salicylates have been found to cause hearing loss and tinnitus, symptoms, which subside after cessation of treatment (Canlon et al., 1998; Chen et al., 2007; Kimitsuki et al., 2009). It is thought that the mechanism of salicylate ototoxicity is similar to that of NSAIDs, that is, a decrease in blood flow to the cochlea (Kimitsuki et al., 2009) or through a

change in the hair cell membrane permeability (Stypulkowski, 1990; Cazals, 2000). Salicylates are the most commonly consumed analgesic, anti-inflammatory and antipyretic drug worldwide (Marchese-Ragonaa, Marionia, Marsonb, Martinic, & Staffieria, 2008; Kimitsuki et al., 2009) and is commonly used in the treatment of cardiovascular disease.

The mechanisms underlying the ototoxic effects of acetaminophen are currently unknown (Yorgason, Kalinec, Luxford, Warren, & Kalinec, 2010). However, it is thought that acetaminophen, which is known to deplete level of glutathione in the body thus causing impaired renal function, may also deplete endogenous cochlea glutathione making the cochlear more susceptible to noise-induced auditory impairment (Curhan et al., 2010). Research by Yorgason et al (2010) showed that high doses of acetaminophen caused inner and outer hair cell death in mice.

A longitudinal study by Curhan et al. (2010) of a group of male Health Professionals (n=26,917), including dentists, optometrists, osteopaths, pharmacists, podiatrists, and veterinarians, aged 40-75 years, found that participants who had regularly used aspirin for 1-4 years were 28% more likely to develop hearing loss than those who did not use aspirin regularly. This study also found a correlation between the degree of hearing loss and the duration of regular use of NSAIDs and acetaminophen. Participants who used either NSAIDs or acetaminophen regularly for 4 years or more were 33% more likely to develop hearing loss than those not regularly taking the pharmaceuticals (Curhan et al., 2010). It was also noted in this study that the association between hearing loss and concomitant use of two or more classes of analgesic appeared to be approximately additive.

A.6.2.3 Radiation

Ionizing radiation is high-frequency radiation that has enough energy to remove an electron from an atom or molecule. It is used in radiographic imaging (also known as “x-rays”), to aid in pathology diagnosis, and therapeutically (radiation therapy) in the treatment of benign and malignant tumours. In dentistry, detailed x-rays enable the clinician to check the state of the client’s teeth and jawbone and therefore, aids in diagnosis dental pathology. The use of radiographic imaging in the clinic predisposes dentist to the side effects of radiation exposure (Ayatollahi et al., 2012).

Common side effects of radiation therapy are fatigue, gastro-intestinal disturbances, skin reactions and localized inflammation depending on the site of the lesion. Sensorineural hearing losses (SNHL) have been reported in patients undergoing head and neck irradiation (Kashiwamura, Fukada, Chida, Satoh, & Inuyama, 2001). Nicholls et al. (1996) reported a 24% incidence of SNHL in patients being treated for nasopharyngeal carcinoma (Nicholls, Chua, Chiu, & Kwong, 1996). The SNHL is often progressive and occurs at the time of irradiation or may develop at a later time (Kashiwamura et al., 2001).

Otological problems that are associated with radiation therapy include Eustachian tube dysfunction, otitis media with effusion, chronic otitis media and conductive or SNHL (Young & Lu, 2001). It is thought that Damage results from ischaemia due to an inflammatory response in the cochlea, organ of Corti and endolymph (Karlidag et al., 2004).

Most studies that have been conducted on the otological effects of exposure to ionizing radiation have focused on sequelae resulting from the high doses dispensed during radiation therapy. Karlidag et al. (2004)

however, look at the effect on hearing of 57 workers exposed to low-dose radiation over a long period and compared them to a control group of unexposed workers (n=32). All those who were exposed to the low-dose radiation worked in a hospital radiology department. The audiometric results obtained showed statistically significant differences in mean thresholds at 4, 8, 10, 12, 14, and 16 kHz between the two groups. As well as a correlation between the duration of exposure to low-doses of radiation and the degree of hearing loss, the study also revealed a statistically significant difference in prevalence of tinnitus and vestibular symptoms between the two groups (Karlidag et al., 2004).

A.6.2.4 Mercury

Mercury and its derivatives have been used for more than thousands of years in medical, chemical, metallurgical and electrical applications. It has been used medically in such applications as antiseptics, antiparasitics, antisphyliotics, and antipruritics and as a diuretic agent, and more recently it has been used in dental amalgams (Kostyniak, 1998; Ozuah, 2000).

Mercury, which is the most common cause of metal poisoning is found in three forms: as elemental or metallic mercury, salts of mercury, such as mercury sulfide or cinnabar, which is used to make red tattoo ink, and organic alkyl-mercurials such as the environmental contaminant methylmercury (Kostyniak, 1998). It is in its elemental state, that mercury is used in dental amalgams (Ozuah, 2000).

The Cawcours brothers, a couple of New York dentists, first used Mercury amalgams in 1883 as cheap and painless treatment for dental caries.

The treatment, which was banned a ten years later by the American Society of Dental surgeons, was considered inexpensive and painless, as it did not require the removal of decay. The current practice of using mercury amalgams began during the twentieth century with the approval from the American Dental Society and the US Bureau of Standards (Ozuah, 2000).

Dental amalgams use mercury in its elemental state, which is volatile at room temperature and when exposed to oxygen readily oxidizes to form mercuric mercury (Kostyniak, 1998). Toxicity is caused by the inhalation of mercury vapour and is often due to improper handling, accidental spills and poor ventilation in the work environment. If not dealt with properly, a spill of elemental mercury can lead to chronic vapour exposure for several weeks to months (Ozuah, 2000).

Mercury vapour is released while chewing and although dental amalgams contain up to 50% elemental mercury, the patient is exposed to approximately 1% of the occupational safe level. Although there is some controversy concerning anecdotal evidence of debilitating side effects, a level of 1% is generally considered to be safe (Ozuah, 2000). Although patients may not be at risk of mercury toxicity, continuous occupational exposure to mercury vapour may be hazardous to the dental practitioner (Kostyniak, 1998).

Ritchie et al. (2001) found a highly significant difference between urinary mercury concentrations of dentists and controls. He reported that the mean concentration of urinary mercury was 4.17 times that of the control group. In this study (n=180), 68% of dental surgeries showed mercury vapour levels above the OSHA occupational exposure standard of 0.05 mg/m³.

Because mercury vapour is essentially odorless and has limited warning properties, workers are often unaware that significant exposure is occurring. When inhaled, 80% of the metallic mercury is absorbed then is rapidly diffused across cell membranes (Kostyniak, 1998; Ozuah, 2000; Ritchie et al., 2001). Exposure to high doses of mercury vapour can lead to biological and neurological injury (Ayatohalli et al., 2012).

Chronic exposure to low doses of elemental mercury leads to central nervous system dysfunction. Once absorbed by the body, elemental mercury has a half life of approximately 30-60 days (Kostyniak, 1998; Ozuah, 2000) and is excreted mostly by the kidneys with small amounts being excreted in other bodily fluids (Ozuah, 2000).

The best treatment for mercury toxicity is prevention (Ozuah, 2000). Efforts to reduce the exposure of dentists to mercury have lead to safer storage and careful handling of metallic mercury. The use of water-cooled drills, improvements in mercury hygiene and ventilation systems, and the use of automated methods of amalgam preparations have lessened the potential risk of mercury vapour exposure to the dental practitioner and support staff (Kostyniak, 1998; Ritchie et al., 2001; Ayatohalli et al., 2012).

A6.3 References

- Albera, R., Lacilla, M., Piumetto, E., & Canale, A. (2009). Noise-induced hearing loss evolution: influence of age and exposure to noise. *European Archives of Oto-rhino-laryngology*, 267, 665-671.
- Attias, J., Bresloff, I., Joachims, Z., & Ising, H. (1998). Prophylactic effect of magesium in noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 271-279). London: Whurr Publishers Ltd.
- Ayatohalli, J., Ayatohalli, F., Mallet Ardekani, A., Barhrololoomi, R., Ayatohalli, J., Ayatohalli, A. (2012). Occupational hazards to dental staff. *Dental Research Journal*, 9(1), 2-7.
- Babisch, W. (2003). Stress hormones in the research on cardiovascular effects of noise. *Noise & Health*, 5(18), 1-11.
- Babisch, W. (2011). Cardiovascular effects of noise. *Noise & Health*, 13(52), 201.
- Barek, S., Adam, O., & Motsch, J. F. (1999). Large band spectral analysis and harmful risks of dental turbines. *Clinical Oral Investigation*, 3, 59-54.
- Bhat, P., Jyothi, C., Kadanakuppe, S., & Ramegowda, C. (2011). Assessment of noise levels of the equipments used in the dental teaching institution, Bangalore. *Indian Journal of Dental Research*, 223, 424-431.
- Bies, D. A., & Hansen, C. H. (1988). *Engineering Noise Control*. London: Unwin Hyman Ltd.
- Broadbent, D. E. (1979). Human performance and noise. In C. M. Harris (Ed.), *Handbook of noise control*. New York: McGraw-Hill, Inc.
- Canlon, B., Fransson, A., & Dagli, S. (1998). Modulating auditory sensitivity to noise trauma by sound conditioning. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Bioogical Effects of Noise* (Vol. 1, pp. 43-51). London: Whurr Publishers Ltd.
- Carmen, R. (1999). Tinnitus and hearing loss. *Occupational Health and Safety*, 68(10), 154-158.
- Carter, L., Gilliver, M., Macoun, D., Rosen, J., & Williams, W. (2012). Music to whose ears? The effect of social norms on young people's risk perceptions of hearing damage resulting from their music listening behaviour. *Noise and Health*, 14(57), 47.
- Cazals, Y. (2000). Auditory sensori-neural alterations induced by salicylate *Progress in Neurobiology* 62, 583-631.
- Chen, Y., Huang, W. G., Zha, D. J., Qiu, J.-H., Wang, J.-L., Sha, S.-H. (2007). Aspirin attenuates gentamicin ototoxicity: from the laboratory to the clinic. *Hearing Research*, 226, 178-182.
- Curhan, S. G., Roland, E., Shargorodsky, J., & Curhan, G. C. (2010). Analgesic use and the risk of hearing loss in men. *American Journal of Medicine*, 123(3), 231-237.
- Damen, G. W. J. A., Pennings, R. J. E., Snik, A. F. M., & Mylanus, E. A. M. (2006). Quality of Life and Cochlear Implantation in Usher Syndrome Type I. *The Laryngoscope*, 116(5), 723-728.
- Davison, S. P., & Marion, M. S. (1998). Sensorineural hearing loss caused by NSAID-induced aseptic meningitis. *Ear, Nose & Throat Journal*, 77(10), 820-821.

- Fernandes, C. S., Carvalho, A. P. O., Gallas, M., Vaz, P., & Matos, P. A. (2006). Noise levels in dental schools. *European Journal of Dental Education*, 28, 32-37.
- Feston, J. M., George, E. L. J., Goverts, S. T., & Hougast, T. (2010). Measuring the effects of reverberation and noise on sentence intelligibility for hearing impaired listeners. *Journal of Speech, Language, and Hearing Research* (Vol. 53, pp. 1429).
- Feuerstein, J., & Marshall, C. (2009). Noise exposure and issues in hearing conservation. In J. Katz, L. Medwetsky, R. Burkard & L. Hood (Eds.), *Handbook of Clinical Audiology*. Baltimore: Lippincott Williams & Wilkins.
- Fidell, S. (1979). Community Response to Noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.
- Gastmeier, W., & Aitken, D. R. (1999). Reverberation in Gymnasia. *Canadian Acoustics*, 27(4), 3-7.
- Gijbels, F., Jacobs, R., Princen, K., Nackaerts, O., & Debruyne, F. (2006). Potential occupational health problems for dentists in Flanders, Belgium. *Clinical Oral Investigation*, 10, 8-16.
- Glorig, A., & Nixon, J. (1962). Hearing loss as a function of age. *Laryngoscope*, 72, 1590-1610.
- Haller, A. K., & Monygomery, J. K. (2004). Noise-induced hearing loss in children. *Teaching Exceptional Children*, 36(4), 22-27.
- Hansen, C. (2005). *Noise Control from Concept to Application*. London: Taylor & Francis.
- Harris, C. M. (1979). Sound and sound levels. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill Inc.
- Harris, C. M. (1991). *Handbook of Acoustical Measurements and Noise Control* (3rd ed.). New York: McGraw-Hill.
- Henderson, D., Subramanian, M., & Boettcher, F. A. (1993). Individual susceptibility to noise-induced hearing loss: an old topic revisited. *Ear & Health*, 14(3), 152-168.
- Hoffman, D. W., Whitworth, C. A., Jones, K. L., & Rybak, L. P. (1987). Nutritional status, glutathione levels, and ototoxicity of loop diuretics and aminoglycoside antibiotics. *Hearing Research*, 31(3), 217-222.
- Holmes, G. B., Goodman, K. L., Hang, D. W., & McCorvey, V. M. (1996). Noise levels in orthopedic instruments and their potential health risks. *Orthopedics* 19(1), 35-37.
- Hood, L. J. (1998). *Clinical Applications of the Auditory Brainstem Response*. New York: Delmar Cengage Learning.
- Iannace, G., Lembo, P., Maffei, L., & Nataletti, P. (2006). *Acoustical conditions and noise exposure inside school gymnasia and swimming pools*. Paper presented at the Euronoise, Tempra: Finland.
- Ising, H., Babisch, W., & Kruppa, B. (1999). Noise-induced endocrine effects and cardiovascular risk. *Noise & Health*, 1(4), 37-48.
- Job, R. F. S. (1996). The influence of subjective reactions to noise on the health effects of the noise. *Environmental International*, 22(1), 93-104.
- Kamal, S. A. (1982). Orthopaedic theatres: a possible noise hazard? *The Journal of Laryngology and Otology*, 96, 985-990.
- Karlıdag, T., Kaygusuz, I., Keles, E., Yalcin, S., Serhatlioglu, S. S., Acik, Y. (2004). Hearing in workers exposed to low-dose radiation for a long period. *Hearing Research*, 194, 60-64.

- Kashiwamura, M., Fukada, S., Chida, E., Satoh, N., & Inuyama, Y. (2001). Sensorineural hearing loss induced by radiation as a late effect: five cases followed by audiogram. *Auris Nasus Larynx*, 28, 111-115.
- Katsuhisa, I., Takeshi, O., Hiroshi, H., & Tomonori, T. (1997). Molecular and clinical implications of loop diuretic ototoxicity. *Hearing Research*, 107(1-2), 1-8.
- Kimitsuki, T., Kakazu, Y., Matsumoto, N., Noda, T., Komune, N., & Komune, S. (2009). Salicylate-induced morphological changes of isolated inner hair cells and outer hair cells from guinea-pig cochlea. *Auris Nasus Larynx*, 36, 152-156.
- Kircher, D. B. (2003). Noise-induced hearing loss. *Journal of Occupational & Environmental Medicine*, 45(6), 579-581.
- Kircher, D. B., Evenson, E., Dobie, R. A., Rabinowitz, P. M., Crawford, J., Kopke, R. (2012). Occupational noise-induced hearing loss: ACOEM task force on occupational hearing loss. *Journal of Occupational & Environmental Medicine*, 54(1), 106-108.
- Kopke, R. D., Weisskopf, P. A., Boone, J. L., Jackson, R. L., Wester, D. C., Hoffer, M. E. (2000). Reduction of noise-induced hearing loss using L-NAC and salicylate in the chinchilla. *Hearing Research*, 149, 138-146.
- Kostyniak, P. J. (1998). Mercury as a potential hazard for the dental practitioner. *New York State Dental Journal*, 64(4), 40-43.
- Kryter, K. D. (1972). Non-auditory effects of environmental noise. *American Journal of Public Health* 62(3), 389-398.
- Kua, H. W., Lee, S. E., & Mahbub, A. S. (2010). A total building performance approach to evaluating building acoustics performance. *Architectural Science Review*, 53(2), 213.
- Laird, I. (2012). *The epidemiology and prevention of NIHL in New Zealand*. Paper presented at the Symposium on Health and the Environment at Work - the Need for Solutions, Wellington, NZ.
- Landstrom, U., & Akerlund, E. (1995). Exposure levels, tonal components, and noise annoyance in working environments. *Environmental International*, 21(3), 265-275.
- Lipscomb, D. M. (1994). *Hearing Conservation in industry, schools, and the Military*. San Diego, CA: Singular Publishing Group.
- Lookwood, A. H., Salvi, R. J., & Burkard, R. F. (2002). Tinnitus. *The New England Journal of Medicine*, 347(12), 904-910.
- Marchese-Ragona, R., Marioni, G., Marson, P., Martinic, A., & Staffieria, A. (2008). The discovery of salicylate ototoxicity. *Audiology and neurotology*, 13, 34-36.
- Marsh, J. P., Jellicoe, P., Black, B., Monson, R. C., & Clark, T. A. (2011). Noise levels in adult and pediatric cast clinics. *The American Journal of Orthopedics*, 40(7), E122-E124.
- McFadden, D., & Plattsmier, H. S. (1983). Aspirin can potentiate the temporary hearing loss induced by intense sounds. *Hearing Research*, 9, 295-316.
- McFadden, D., Plattsmier, H. S., & Pasanen, E. G. (1984). Temporary hearing loss induced by combinations of intense sounds and nonsteroidal anti-inflammatory drugs. *American Journal of Otolaryngology*, 5, 235-241.
- McKinnon, B. J., & Lassen, L. F. (1998). Case report: Naproxen-associated sudden sensorineural hearing loss. *Military Medicine*, 163(11), 792-793.

- Melnick, W. (1991). Human temporary threshold shift and damage risk. *The Journal of the Acoustical Society of America*, 90(1), 147-153.
- Mervine, R. (2007). Noise-induced hearingloss in dental offices. *Dental Economics*, 97(1), 1-7.
- Messano, G. A., & Petti, S. (2012). General dental practitioners and hearing impairment. *Journal of Dentistry*, 40, 821-828.
- Miller, J. D. (1974). Effects of noise on people. *The Journal of the Acoustical Society of America*, 56, 729-764.
- Molino, J. A. (1979). Annoyance and Noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.
- Nicholls, J. M., Chua, D., Chiu, P. M., & Kwong, D. L. W. (1996). The detection of clinically occult nasopharyngeal carcinoma in patients following radiotherapy – an analysis of 69 patients. *The Journal of Laryngology & Otology*, 110(5), 496-499.
- OSH. (2002). *Approved code of practice for the management of noise in the workplace*. Wellington: Occupational Safety and Health Service.
- Ozuah, P. O. (2000). Mercury poisoning. *Current Problems in Pediatrics*, 30, 91-99.
- Parham, K., Gates, G., Dobie, R. A., McKinnon, R., & Backous, D. (2010). Challenges and opportunities in presbycusis. *Otolaryngology-Head and Neck Surgery*, 143, 31.
- Patel, R., & Schell, K. W. (2008). The influence of linguistic content on the Lombard effect. *Journal of Speech, Language, and Hearing Research*, 51(1), 209-220.
- Peterson, A. P. G. (1979). Noise measurements: instruments. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill, Inc.
- Prasher, D. (1998). Factors influencing susceptibility to noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 125-131). London: Whurr Publishers Ltd.
- Pyykko, I., Starck, J., Toppila, E., & Kaksonen, R. (1998). Ageing as a major confounding factor in noise-induced hearing loss. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 157-163). London: Whurr Publishers Ltd.
- Raney, J. P., & Cawthorn, J. M. (1979). Aircraft noise. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw Hill Inc.
- Ritchie, K. A., Gilmour, W. H., Macdonald, E. B., McGowan, D. A., Dale, I. M., Hammersley, R. (2001). Health and neuropsychological functioning of dentists exposed to mercury. *Occupational and Environmental Medicine*, 59, 287-293.
- Royster, J. D., Royster, L. H., & Killion, M. C. (1991). Sound exposures and hearing thresholds of symphony orchestra musicians. *The Journal of the Acoustical Society of America*, 89(6), 2793-2802.
- Rybak, L. P., & Ramkumar, V. (2007). Ototoxicity. *Kidney International*, 72(8), 931-935.
- Schroeder, M. R. (1980). Acoustics in human communications: Room acoustics, music, and speech. *Journal of the Acoustical Society of America*, 68(1), 22-29.

- Setcos, J. C., & Mahayuddin, A. (1998). Noise levels encountered in dental clinical and laboratory practice. *International Journal of Prosthodontics*, 11, 150-157.
- Sharland, I. (1972). *Woods Practical Guide to Noise Control*. London: Woods of Cholchester Limited.
- Shepard, D., Welch, D., Dirks, K. N., & Mathews, R. (2010). Exploring relationships between noise sensitivity, annoyance and health-related quality of life in a sample of adults exposed to environmental noise. *International Journal of Environmental Research and Public Health*, 7, 3579-3594.
- Siverdeen, Z., Ali, A., Lakdawala, A. S., & McKay, C. (2008). Exposure to noise in orthopaedic theatres – do we need protection? *International Journal of Clinical Practice*, 62(11), 1720-1722.
- Smith, A. (1991). A reveiw of the non-auditory health effects of noise on health. *Work & Noise*, 5(1), 49-62.
- Sorainen, E., & Rytönen, E. (2002). Noise level and ultrasound spectra during burring. *Clinical Oral Investigation*, 6, 133-136.
- Speaks, C. E. (2005). *Introduction to Sound: Acoustics for Hearing and Speech Sciences*. USA: Thomson Delmar Learning.
- Spreng, M. (2000). Possible health effects of noise induced cortisol increase. *Noise & Health*, 2(7), 59-63.
- Stach, B. A. (1998). *Clinical Audiology: an Introduction*. San Diego: Singular Publishing Group, Inc.
- Stach, B. A. (2003). *Comprehensive Dictionary of Audiology Illustrated* (2nd ed.). New York: Delmar Cengage Learning.
- Stansfeld, S. A., & Matheson, M. P. (2003). Noise pollution: non-auditory effects on health. *Bristish Medical Bulletin*, 68, 243-257.
- Starck, J. (1998). How should different susceptibility factors be evaluated. In D. Prasher & L. Luxon (Eds.), *Advances in Noise Research: Biological Effects of Noise* (Vol. 1, pp. 121-123). London: Whurr Publishers Ltd.
- Stypulkowski, P. H. (1990). Mechanisms of salicylate ototoxicity. *Hearing Research*, 46, 113-146.
- Suter, A. H., & Berger, E. H. (2002). *Hearing conservation manual* (4 ed.). Milwaukee.
- Sydney, S. E., Lepp, A. J., Whitehouse, S. L., & Crawford, R. W. (2007). Noise exposure due to orthopedic saws in simulated total knee arthroplasty surgery. *The Journal of Arthroplasty*, 22(8), 1193-1197.
- Thorne, P. R., Ameratunga, S. N., Stewart, J., Reid, N., Williams, W., Purdy, S. C. (2008). Epidemiology of noise-induced hearing loss in New Zealand. *The New Zealand Medical Journal*, 121(1280), 1-9.
- Trenter, S. C., & Walmsley, A. D. (2003). Ultrasonic dental scaler: associated hazards. *Journal of Clinical Periodontology*, 30, 95-101.
- van Dijk, F. J. H. (1990). Epidemiological research on non-auditory effects of occupational noise exposure. *Environmental International*, 16, 405-409.
- Venema, T. H. (2006). *Compression for Clinicians* (2nd ed.). Canada: Thompson Delmar Learning.
- Ward, W. D. (1971). Presbycusis, sociocusis and occupational noise-induced hearing loss. *Proceedings of the Royal College of Medicine*, 64, 200-203.
- Webster, J. C. (1979). Effects of Noise on Speech. In C. M. Harris (Ed.), *Handbook of Noise Control*. New York: McGraw-Hill, Inc.

- WHO. (1997). Prevention of noise-induced hearing loss. Geneva: World Health Organisation.
- WHO. (1999). Constitution of the World Health Organization
- Willett, K. M. (1991). Noise-induced hearing loss in orthopaedic staff. *The Journal of Bone and Joint Surgery*, 73 B(1), 113-115.
- Williams, W., & Purnell, J. (2010). The statistical distribution of expected noise level output from commonly available personal stereo players. *Acoustics Australia*, 38, 119-122.
- Willich, S. N., Wegscheider, K., Stallmann, M., & Keil, T. (2006). Noise burden and the risk of myocardial infarction *European Heart Journal*, 27, 276-282.
- Yorgason, J. D., Kalinec, G. M., Luxford, W. M., Warren, F. M., & Kalinec, F. (2010). Acetaminophen ototoxicity after acetaminophen/ hydrocodone abuse: Evidence from two parallel in vitro mouse models. *Otolaryngology-Head and Neck Surgery* 142, 814-819.
- Yost, W. A. (2000). *Fundamentals of Hearing: An Introduction* (4th ed.). San Diego: Academic Press, Inc.
- Yost, W. A., & Neilson, D. W. (1997). *Fundamentals of Hearing: An Introduction*. USA: Holt, Rinehart and Winston.
- Young, Y. H., & Lu, Y. C. (2001). Mechanism of hearing loss in irradiated ears: a longitudinal study. *Annals Otology Rhinology Laryngology*, 110, 904-906.
- Zollinger, S. A., & Brumm, H. (2011). The Lombard effect. *Current Biology*, 21(16), 614-615.
- Zubick, H. H., Tolentino, A. T., & Boffa, J. (1980). Hearing loss and the highspeed dental drill. *American Journal of Public Health*, 70(6), 633-635.

A.7

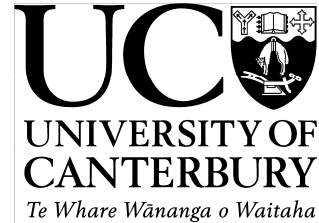
Appendix 7 – Participant Information

A.7.1 Summary

The following section contains introductory letters, background information and consent forms that were used in this study.

A.7.2 Letter to Employers: Dental

Date



Dear

I am a Master of Audiology student at the University of Canterbury. As part of my Audiology Masters I am intending to investigate noise levels in dental surgeries.

There is anecdotal evidence that those working in dental clinics suffer from noise-induced hearing-loss; my research will be looking to find evidence that may support this. The research will involve recording sound levels produced by dental equipment and analyze the measurements.

I am hoping that you, as the owner of XXXX dental surgery, will be happy for your staff to participate in my research. My supervisor for this research will be Dr. John Pearce, Department of Mechanical Engineering, University of Canterbury, along with Dr. Don Sinex, Department of Communication Disorders, University of Canterbury.

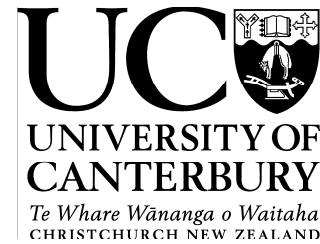
Please be assured that all information and results obtained will remain anonymous and confidential and that no information will be given to any third party. On completion of my research, and subsequent Thesis, I will be happy to send you a summary of my results. An outline of the research can be found over the page.

If you have any queries with regards to the research please contact me. I may be contacted on my cellphone 027 XXX XXXX or you may email me at cfc14@uclive.ac.nz

Regards
Carol Crowther

A.7.3 Letter to Employers: Orthopaedic

Date



Orthopaedics Outpatients Department,
Christchurch Public Hospital.

Dear Charge Nurse,

I am a Master of Audiology student at the University of Canterbury. As part of my Audiology Masters I am intending to investigate noise levels found in orthopaedic environments.

There is anecdotal evidence that those working in orthopaedic clinics suffer from noise-induced hearing-loss, my research will be looking to find evidence that may support this. The research will involve recording sound levels produced by orthopaedic equipment and analyse the measurements.

I am hoping that you and your nurses working in the plaster-room would be willing to participate in my research. My supervisor for this research will be Dr. John Pearce, Department of Mechanical Engineering, University of Canterbury, along with Dr. Don Sinex, Department of Communication Disorders, University of Canterbury.

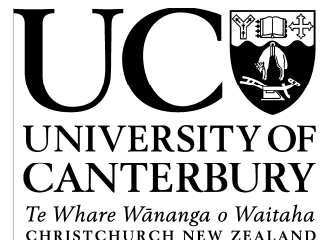
Please be assured that all information and results obtained will remain anonymous and confidential and that no information will be given to any third party. On completion of my research, and subsequent Thesis, I will be happy to send you a summary of my results. An outline of the research can be found over the page.

If you have any queries with regards to the research please give me a call. I may be contacted on my cellphone 027 XXX XXXX or you may email me at cfc14@uclive.ac.nz

Regards
Carol Crowther

A.7.5 Consent Form

University of Canterbury
 Department of Communication Disorders
 Private Bag 4800
 Christchurch 8140
 New Zealand



Researcher: Carol Crowther

Contact address: University of Canterbury
 Department of Communication Disorders
 Private Bag 4800
 Christchurch 8140
 New Zealand

Date: 14 May 2012

Consent Form

“Noise Levels in the Health Industry in New Zealand”

I have read and understood the description of the above-named project. On this basis, I agree to participate as a subject in the project, and I consent to publication of the results of the project with the understanding that anonymity will be preserved.

I provide my consent to be recorded.

I understand also that I may at any time withdraw from the project, including withdrawal of any information I have provided.

I note that the project has been reviewed **and approved** by the University of Canterbury Human Ethics Committee.

Name: (please print):

Signature: -

Date:
